JUN 14 1944

Volume 23 Number 2

JOURNAL of DENTAL RESEARCH

FOUNDED BY WILLIAM J. GIES IN 1919

Official Publication of the

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

April, 1944

The Effect of Proteolytic Enzymes on Acid Formation in the Mouth. *L. S. Fosdick and G. W. Rapp* (Northwestern University) 81

The Effect of Amylolytic Enzymes on Acid Production in Saliva. *L. S. Fosdick and G. W. Rapp* (Northwestern University) 85

Studies on the Incidence and Cause of Dental Defects in Children. VI. Pits and Fissures. *Marcu Brucker, D.D.S.* (Board of Education, Newark, N. J.) 89

Studies on the Incidence and Cause of Dental Defects in Children. VII. Fissures and Caries. *Marcu Brucker, D.D.S.* (Board of Education, Newark, New Jersey) 101

A Study of the Bilateral Incidence of Carious Lesions. *David B. Scott, D.D.S.* (University of Rochester and University of Maryland) 105

A Critical Microscopic Technic Applied to Study of Human Dentin. *Wardlaw M. Hammond, Lester W. Burkett, D.D.S., M.D., and Seymour J. Kreshover, D.D.S., Ph.D.* (University of Pennsylvania and Yale University) 111

A Study of the Pattern and Combinations of Congenitally Missing Teeth in Man. *Peter J. Brekhus, Clarence P. Oliver and George Montelius* (University of Minnesota) 117

Congenital Partial Hemihypertrophy Involving Marked Malocclusion. *Charles E. Rudolph, D.D.S., and Rudolph W. Norvold, D.D.S., M.S.D.* (University of Minnesota) 133

Dental Caries. *B. Gottlieb* (Baylor University) 141

International Association for Dental Research, Proceedings of New York Section, Jan. 27, 1944, compiled by *Frances Krasnow* 151

PUBLISHED BI-MONTHLY AT MT. ROYAL AND GUILFORD AVENUES, BALTIMORE 2, MD.
By THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

EDITORIAL OFFICE: 4559 Scott Ave., St. Louis, Mo.

Copyright, 1944, by the International Association for Dental Research

Made in United States of America

THIS JOURNAL IS MANUFACTURED UNDER WARTIME CONDITIONS IN CONFORMITY WITH ALL GOVERNMENT REGULATIONS CONTROLLING THE USE OF PAPER AND OTHER MATERIALS

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

EDITORS OF THE JOURNAL OF DENTAL RESEARCH

Editor Emeritus—William J. Gies

Publication Committee: J. Frank Hall, S. S. Arnim and Hamilton B. G. Robinson.

Editor—Hamilton B. G. Robinson. *Business Manager*—J. Frank Hall. *Circulation Manager*—S. S. Arnim.

Assistants to the editor—L. R. Boling and W. E. Koch, Jr. *Assistant to the business manager*—R. L. Simpson, Jr. *Assistant circulation manager*—Virgil D. Cheyne.

Board of Editors—Editor-emeritus, members of the Publication Committee and sectional representatives named in fifth column below.

THE SECTIONS—THEIR SECRETARIES, AND REPRESENTATIVES IN THE COUNCIL AND IN THE BOARD OF EDITORS (1941)

Division	*Section*	*Secretary*	*Councillor*	*Editor*
Canada	Halifax	S. G. Ritchie	J. S. Bagnall	S. G. Ritchie
	Toronto	A. W. Ellis	A. D. Mason	Thos. Cowling
	Winnipeg	E. Roy Bier	H. J. Merkeley	E. Roy Bier
China	Chengtu	R. Gordon Agnew	R. Gordon Agnew	R. Gordon Agnew
England	London	Evelyn Sprawson	Evelyn Sprawson	Evelyn Sprawson
Germany	Prague	Friedr. Neumann	Friedr. Neumann	Jan Jesensky
	Vienna	Fritz Driak	R. Trauner	F. Shönbauer
Hungary	Budapest	Ferenc Köszeg	———	———
Palestine	Palestine	S. Lewin-Epstein	S. Lewin-Epstein	S. Lewin-Epstein
South Africa	Johannesburg	Julius Staz	J. N. Noriskin	W. B. Osborn
United States	Ann Arbor	Philip Jay	Philip Jay	Philip Jay
	Baltimore	M. S. Aisenberg	M. S. Aisenberg	J. B. Robinson
	Boston	G. E. Thompson	L. M. S. Miner	P. E. Boyle
	Chicago	M. K. Hine	A. G. Brodie	L. S. Fosdick
	Cleveland	S. W. Chase	Thomas J. Hill	S. W. Chase
	Columbus	R. D. McFarland	J. H. Kaiser	H. D. Spangenberg
	Detroit	Samuel J. Lewis	P. C. Lowery	Samuel J. Lewis
	Houston	E. Kotanyi	E. Kotanyi	E. Kotanyi
	Indianapolis	Hannah E. Silberstein	V. D. Cheyne	T. D. Speidel
	Iowa City	A. O. Klaffenbach	A. W. Bryan	George S. Easton
	Louisville	Raymond E. Myers	J. T. O'Rourke	H. T. Knighton
	Minnesota	W. J. Simon	P. J. Brekhus	W. D. Armstrong
	New Orleans	C. S. Tuller	S. L. Tiblier	F. J. Wolfe
	New York	Isaac Neuwirth	L. M. Waugh	Frances Krasnow
	Philadelphia	Thomas J. Cook	J. L. T. Appleton	J. L. T. Appleton
	Pittsburgh	L. E. Van Kirk	H. E. Friesell	L. E. Van Kirk
	Richmond	A. P. Little	S. S. Arnim	Harry Lyons
	Rochester	H. B. McCauley	H. B. McCauley	H. B. McCauley
	San Francisco	A. J. Ker	W. C. Fleming	Herman Becks
	St. Louis	R. C. Wheeler	W. E. Koch, Jr.	R. F. Bleiker
	Washington	H. W. Krogh	F. S. Arnold	C. A. Schlack
Materials Group		E. W. Skinner	W. S. Crowell	I. C. Schoonover

OFFICERS OF THE ASSOCIATION

President—H. Trendley Dean, *President-elect*—Wallace D. Armstrong, *Vice-president*—Samuel Chase, *Secretary-treasurer*—Edward H. Hatton, *Editor*—Hamilton B. G. Robinson, *Trustees*—R. W. Bunting, Edward H. Hatton, Thomas J. Hill, Leuman M. Waugh, J. L. T. Appleton.

Subscription price $5.00 per volume. Single numbers when available $1.00.

Entered as second-class matter June 24, 1919, at the Post Office at Baltimore, Maryland, under the act of March 8, 1879

WAVERLY PRESS, INC.

BALTIMORE, U. S. A.

THE EFFECT OF PROTEOLYTIC ENZYMES ON ACID FORMATION IN THE MOUTH[1]

L. S. FOSDICK AND G. W. RAPP

Chemistry Department, Northwestern University Dental School, Chicago, Ill.

It has been known for many years that fermentation is caused by the chemical action of enzymes rather than by the living organism, and that the enzymes are active even when the living organism which produces the enzyme is dead (1). It is now known that the production of acids in the mouth is also the result of enzyme action (2, 3) and that the mechanism involved is no exception to the general mechanism of fermentation and carbohydrate metabolism (4, 5). Most of the enzymes that have been isolated and identified are protein in nature (6, 7, 8, 9, 10, 11, 12) and if this is the case, it should be possible to inactivate them by means of substances that destroy the protein molecule. Thus, it might be possible to inhibit acid formation in saliva by the use of a proteolytic enzyme which would act on proteins under the normal conditions that exist in saliva. Among the proteolytic enzymes that are commercially available and are active in the reaction that prevails in normal saliva are papain, trypsin, and pancreatin. The effect of these enzymes in various concentrations is described in this paper.

EXPERIMENTAL PROCEDURE

Saliva was gathered by paraffin stimulation, well mixed, and divided into several different portions of 10 cc. each. One sample of the fresh saliva was analyzed for calcium. To each of the other samples were added 1 gram of glucose and 0.1 gram of 300 mesh powdered human tooth enamel. In addition to this, appropriate amounts of the test material were added to the samples, with the exception of 2 that were used as controls. Two samples of each concentration were made up in order to secure checks. The samples were then sealed and placed in a water bath and incubated at 37° C. with constant agitation. This constant shaking is essential in order to facilitate the solution of the enamel as fast as the acids are formed. After 4 hours of incubation the samples were removed and analyzed for calcium. This procedure is essentially the same as the chemical test for caries susceptibility (13). The acids formed were expressed as milligrams of calcium dissolved per 100 cc. of saliva. The results are in the Table I.

DISCUSSION

Contrary to expectations, the proteolytic enzymes did not cause an inhibition of acid formation. Trypsin and papain caused a marked stimulation, while

[1] This work was done under a grant from The Abbott Foundation for Medical Research.
Received for publication December 8, 1943.

pancreatin showed no appreciable effect. It is entirely possible that the protein of the enzymes concerned with carbohydrate degradation is more resistant to the action of proteolytic enzymes than is the normal salivary protein. No explanation as to the cause of the stimulation is offered.

TABLE I

The effect of proteolytic enzymes on acid production

TUBE	MG. ENZYME PER 100 CC.	MG. CALICUM PER 100 CC. SALVIA	
TRYPSIN			
0	control saliva before incubation	4.5	4.5
1	0	12.8	13.5
2	0	13.0	13.1
3	10	13.2	13.2
4	20	14.4	14.3
5	40	16.7	16.9
6	80	17.8	17.4
7	100	21.8	22.4
8	200	24.5	25.6
PANCREATIN			
0	control saliva before incubation	5.7	5.5
1	0	22.2	23.0
2	0	22.5	22.9
3	10	22.8	22.8
4	20	23.5	23.6
5	30	23.9	23.9
6	60	25.4	25.5
7	100	24.5	24.2
8	200	21.1	20.4
PAPAIN			
0	control saliva before incubation	5.5	5.4
1	0	13.2	13.2
2	0	13.0	13.2
3	14	13.4	13.8
4	28	14.1	14.2
5	56	15.4	15.5
6	112	16.0	17.9
7	224	17.9	18.1
8	448	22.5	23.5

SUMMARY

The effect of papain, trypsin, and pancreatin on the rate of acid formation in saliva has been studied. The trypsin and papain stimulate acid formation, while the pancreatin has no effect.

REFERENCES

1. Buchner, E., *Ber. Deutsch. Chem. Gesellsch.*, **31**, 568, 1898.
2. Fosdick, L. S., and Hansen, H. L., *J. A. D. A.*, **23**, 401, 1936.

3. Fosdick, L. S., *J. A. D. A.*, **26**, 415, 1939.
4. Parnas, I. K., *Klin. Wochenschr.*, **14**, 1017, 1935.
5. Wood, H. G., Stone, R. W., and Werkman, C. H., *Biochem. J.*, **31**, 349, 1937.
6. Werkman, C. H., and Wood, H. G., *Botanical Rev.*, **8**, 1, 1942.
7. Northrop, J. H., and Kunitz, M., *J. Gen. Physiol.*, **16**, 267, 1932.
8. Northrop, J. H., *J. Gen. Physiol.*, **13**, 739, 1930.
9. Northrop, J. H., and Kunitz, M., *J. Gen. Physiol.*, **18**, 433, 1935.
10. Theorell, H., *Biochem. Z.*, **278**, 263, 1935.
11. Anson, M. L., *Science*, **81**, 467, 1935.
12. Sumner, J. B., and Dounce, A. L., *J. Biol. Chem.*, **121**, 417, 1937.
13. Fosdick, L. S., Hansen, H. L., and Epple, C., *J. A. D. A.*, **24**, 1275, 1937.

THE EFFECT OF AMYLOLYTIC ENZYMES ON ACID PRODUCTION IN SALIVA[1]

L. S. FOSDICK AND G. W. RAPP

Chemistry Department, Northwestern University Dental School, Chicago, Ill.

It has been shown that pancreatic amylase inhibits the formation of lactic acid from glycogen and starch by muscle enzymes. It was postulated that this action is due to an inhibition of the formation of hexose phosphates (1, 2, 3). Although much confusion still exists concerning the first steps in the conversion of glycogen to lactic acid, it is quite probable that they involve the formation of a hexose phosphate (4). It has been shown that the production of lactic acid in saliva under the influence of bacterial enzymes requires the same types of reaction as are involved in the conversion of the hexose phosphate to lactic acid in muscle tissue and the conversion of monosaccharides to lactic and other acids by bacterial enzymes (5, 6, 7, 8). Although it was shown that amylase did not inhibit acid formation from glucose as it did with glycogen using muscle enzymes, it was thought that some effect might be observed when bacterial enzymes were used. For this reason it was thought interesting to study the effect of amylolytic enzymes on acid formation in saliva. Furthermore, it was thought that a study of this nature might explain some of the observations concerning the effect of ptyalin on dental caries (9, 10, 11). This paper deals with the effect of pancreatic and salivary amylase on the rate of acid production in saliva.

EXPERIMENTAL PROCEDURE

Saliva was gathered by paraffin stimulation, well mixed, and divided into several different portions of 10 cc. each. One sample of the fresh saliva was analyzed for calcium. To each of the other samples was added 1 gram of glucose and 0.1 gram of 300 mesh powdered human tooth enamel. In addition to this, appropriate amounts of the test material were added to the samples, with the exception of 2 that were used as controls. Two samples of each concentration were made up in order to secure checks. The samples were then sealed and placed in a water bath and incubated at 37° C., with constant agitation. This constant shaking is essential in order to facilitate the solution of the enamel as fast as the acids are formed. After 4 hours of incubation the samples were removed and analyzed for calcium. This procedure is essentially the same as the chemical test for caries susceptibility (12). The acids formed were expressed as milligrams of calcium dissolved per 100 cc. of saliva.

As ptyalin in pure or concentrated form could not be obtained, the above technique could not be employed with this enzyme. Saliva was gathered from

[1] This work was done under a grant from The Abbott Foundation for Medical Research. Received for publication December 8, 1943.

47 individuals by paraffin stimulation. A portion of each sample was analyzed for ptyalin activity by the method of Meyers (13) and the rest of the sample was used for determining the rate of acid formation. Glucose and powdered

TABLE I

The effect of amylopsin on acid production

TUBE	MG. AMYLOPSIN PER 100 CC.	MG. CALCIUM PER 100 CC. SALIVA	
0	control saliva before incubation	4.8	4.6
1	0	13.2	13.2
2	0	13.0	13.2
3	14	13.4	13.8
4	28	14.1	14.2
5	56	15.4	15.5
6	112	16.0	17.9
7	224	17.9	18.1
8	448	22.5	23.5

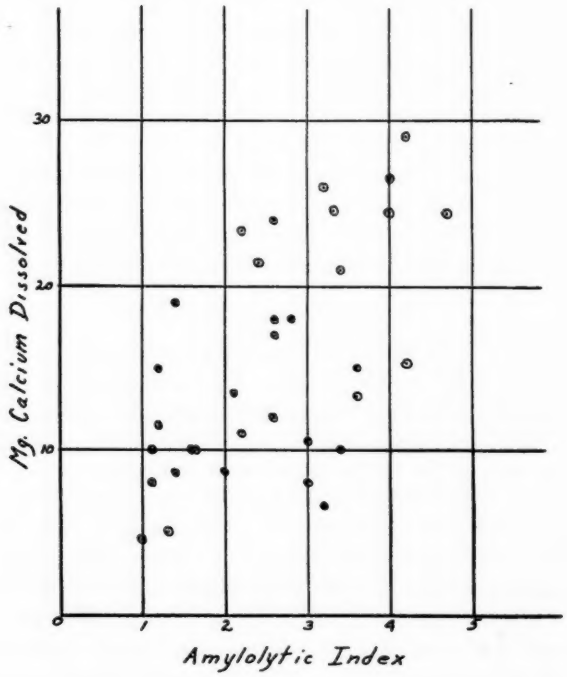


FIG. 1

enamel were added and the sample was allowed to incubate at body temperature; the rate of acid formation was determined in terms of calcium dissolved, as previously described. The rate of acid formation was plotted against the ptyalin index. The results are recorded in the Table I and *fig. 1.*

DISCUSSION

The results were quite surprising, as instead of an inhibition, as was observed in the formation of lactic acid from glycogen, there was a marked stimulation when pancreatic amylase was used. The stimulation was not appreciable in the lower concentrations, but in concentrations of 400 mg./100 cc. there was a 100% increase in the rate of acid production. The stimulation can not be attributed to the amylolytic activity as such, as the sugar used was glucose, which is unaffected by amylase. No explanation is offered for this stimulation however, it is entirely possible that it affects the enzyme systems involved in the degradation of monosaccharides.

When the rate of acid formation was plotted against the ptyalin index, a similar result was obtained. In general, the salivary samples that contained the largest amount of ptyalin formed acid the fastest. This stimulating effect was not constant, however, as many samples with a high ptyalin index formed acid slowly, and many samples with a low ptyalin index formed acid rapidly. This would indicate that there are other factors in saliva which play a major role in acid production.

SUMMARY

The effect of pancreatic amylase on acid production has been studied. It was found that pancreatic amylase stimulates acid production.

The ptyalin index of saliva was compared with the rate of acid production in 47 samples of saliva. There was a general trend toward faster acid production in samples with a high ptyalin index, but there were many exceptions to this trend.

REFERENCES

1. Case, E. M., and McCullagh, D. R., *Biochem. J.*, **22**, 1060, 1928.
2. Case, E. M., *Biochem. J.*, **23**, 210, 1929.
3. *Idem.*, *Biochem. J.*, **25**, 561, 1931.
4. Parnas, I. K., *Klin. Wochenschr.*, **14**, 1017, 1935.
5. Fosdick, L. S., and Hansen, H. L., *J. A. D. A.*, **23**, 401, 1936.
6. Fosdick, L. S., *J. A. D. A.*, **26**, 415, 1939.
7. Wood, H. G., Stone, R. W., and Werkman, C. H., *Biochem. J.*, **31**, 349, 1937.
8. Werkman, C. H., and Wood, H. G., *Botanical Rev.*, **8**, 1, 1942.
9. Prinz, H., *D. Cosmos*, **60**, 197, 1918.
10. Purjesz, B., *Wien. Klin. Wochenschr.*, **26**, 1307, 1913.
11. Michel, Dr., *D. Cosmos*, **57**, 943, 1915.
12. Fosdick, L. S., Hansen, H. L., and Epple, C., *J. A. D. A.*, **24**, 1275, 1937.
13. Meyers, V. C., *Laboratory Directions in Biochemistry* (St. Louis, Mosby Co.: 1942).

STUDIES ON THE INCIDENCE AND CAUSE OF DENTAL DEFECTS IN CHILDREN[1]

VI. Pits and Fissures

MARCU BRUCKER, D.D.S.

Dental Division, Bureau of Health Education and Service, Board of Education, Newark, N. J.

One of the most important problems encountered in the study of the incidence of dental defects in children is that of pits and fissures. A fissure may be defined as an anatomical defect caused by failure of the enamel lobes to coalesce perfectly during the formative stages. According to Dunning and Davenport (1) the term "pit" may be used synonymously.

Apparently most dentists have accepted the conclusion of Hyatt (2) that "the chances are over 2,500 to one that decay will occur in a pit or fissure". Undoubtedly Hyatt's belief was inspired by the mathematical study of Bodecker (3) who expressed his conviction that "99 out of every 100 fissures decay". In the same year Bodecker (4) published his resolution: "as 98 per cent of the first permanent molars do become carious, be it resolved to place small fillings in the grooves of these teeth, irrespective of whether they are carious or non-carious, as soon as practicable after eruption."

Bodecker's claims have never been substantiated by clinical follow-up observations carried out by himself or by other investigators, yet they appear to have received the tacit approval of a majority of the profession. Consequently, with Hyatt and Bodecker leading those interested in the "prevention" of caries, a new field was created in prevention procedure—that of prophylactic odontotomy.

The founding of this new procedure of dentistry stimulated an impulse on the part of most practitioners to open every fissure and insert a filling as soon as a molar tooth erupted. Many premolars are similarly treated. That the fissures be carious is not necessary, for Hyatt (5) strongly emphasizes that "if any hesitancy is felt about cutting into the grooves which have fissures at their base but without apparent decay, the operator should remember that 99 out of every 100 of these fissures will have to be filled some day". Hyatt's contention, like that of Bodecker, has never been substantiated by clinical proof and it appears that never can be, yet the response to this teaching was tremendous the world over. In their enthusiasm, those engaged in its operation failed to consider that the cutting away of healthy structure, like the cutting away of diseased tooth substance, is operative not preventive dentistry. Munblatt's (6) warning that the prevention of dental caries is not accomplished by the simple cutting out of teeth for the purpose of correcting a deficiency in their anatomical make-up

[1] Received for publication March 17, 1943. Revised by author June 10, 1943.

received no consideration. His inference that it was not as yet demonstrated that the dental organs are endowed with the quality of tolerating surgical intervention in the absence of actual disease was almost completely ignored. Similarly, A. D. Black's (7) admonishment that the filling of teeth helps to control, rather than prevent, dental decay was accepted by only a few.

As the cause of caries remains an unsolved problem, prophylactic odontotomy received the approbation of practically the entire profession and much has been written in its praise. It has been adopted as a routine practice in clinics and by private practitioners and is now performed in most parts of the world. Assumptions were accepted as facts and an empirical formula was given recognition without verification. At first glance there appears to be evidence in favor of opening all fissures, whether carious or not, but if we proceed with a scientific investigation of the subject, we find that the evidence is only circumstantial. Let us now consider some of the views set forth on the problem under discussion.

In the opinion of Prime (8) pits and fissures of the first molars should be filled. He states, as a result of his own experience, that there is no other way to take care of them. In his practice, he adds, "all pits and fissures in the bicuspids are filled soon after eruption". Harris (9) advocated that fissures be cut out and filled with "silver amalgam". His contention is based on the fact that "95 per cent of the molar teeth *ARE REPORTED* to have these defects upon eruption."[2] I find it difficult to express the impact that such an assertion makes upon me, for I must question the facts. In one of my very recent examinations of a group of 111 children of both sexes, whose ages ranged from 5 to 6 years, it was noted that a total of 167 permanent first molars had erupted. Of this number only 4 disclosed defective fissures; 2 others had caries (Table III). Brekhus (10) considered pits and fissures as congenital faults. He reasons that the profession should agree with Hyatt and overcome these defects by means of prophylactic odontotomy. However, I find it impossible to reject my belief that his views are based on the unusual circumstance that he noted 83,732 filled teeth in the 10,045 students he investigated. It seems to me definitely unproved that all of the filled teeth were previously carious as Brekhus considered them. My view is strenghthened by the implication that all of his subjects "were able to obtain the best that dentistry has to offer". McFall (11) also advocates this measure. In his own words: "Scientific and tested statistical data indicate these surfaces of the teeth most susceptible to dental caries". This may be true, but it must not be forgotten that such adjustments will not eliminate the possibility of a future initiation of caries—and its sequelae—at the margins of these artificial cavities.

Starr (12), who made histological examinations of the depths of fissures, concludes that all deep pits and fissures in newly erupted molars should be subjected to prophylactic odontotomy. He does not explain their failure to develop into caries in many individuals who have long since passed adolescence. Rich (13) regarded the procedure as fundamentally sound and believed that these imper-

[2] Italics are the present author's.

fections should receive immediate care. McCall (14) subscribed to the same view.

In a report compiled from the records of 300 children who attended his department at New York University, Cotton (15) related that at his clinic "every pit and fissure is prepared and filled when a fine explorer can enter and catch". Nevertheless, a "catch" with a fine explorer may not denote the presence of

TABLE I

Incidence of caries of the first permanent molars in boys and girls of distinct age groups

	BOYS				GIRLS			
	Ages			Total	Ages			Total
	6–9	10–12	13–15		6–9	10–12	13–15	
No. of molars studied:								
White	3108	3005	1578	7691	3270	2554	1215	7039
Negro	*557*	*600*	*299*	*1456*	*529*	*459*	*166*	1154
No. of molars carious:								
White	624	1272	893	2789	723	1044	719	2486
Negro	*75*	*199*	*135*	*409*	*82*	*121*	*52*	*255*
% carious:								
White	20.1	42.3	56.5	36.2	22.1	40.8	59.1	35.3
Negro	*13.8*	*33.1*	*45.1*	*28.0*	*15.5*	*26.3*	*31.3*	*23.0*

TABLE II

Susceptibility to caries of each of the 4 first permanent molars in boys and girls

	BOYS				GIRLS			
	Teeth							
	$\underline{6}\vert$	$\vert\underline{6}$	$\overline{6}\vert$	$\vert\overline{6}$	$\underline{6}\vert$	$\vert\underline{6}$	$\overline{6}\vert$	$\vert\overline{6}$
No. of molars studied:								
White	1915	1959	1967	1962	1722	1821	1739	1757
Negro	*367*	*383*	*364*	*374*	*279*	*294*	*296*	*287*
No. of molars carious:								
White	545	575	826	842	475	492	731	782
Negro	*90*	*81*	*116*	*122*	*38*	*43*	*78*	*87*
% carious:								
White	28.4	29.3	41.9	42.8	27.5	27.0	42.0	44.5
Negro	*24.5*	*22.3*	*31.8*	*32.6*	*13.6*	*14.6*	*26.3*	*30.3*

caries. Taking it for granted that caries is present or may develop whenever this instrument "sticks" is a lamentable attitude. Such beliefs should have no place in scientific dentistry. Anderson (16) correctly points out that "the tip of the explorer will readily catch any deep pit or fissure where the enamel is intact, as well as where it is broken by decay". That the tip of the explorer will catch any deep pit or fissures, even though perfectly normal, has been demonstrated to me in thousands of instances. Because of the fact that caries

may develop even in apparently sound enamel structure, McCall (17) has adopted "a policy of filling food retentive fissures even when there is no evidence of imperfect coalescence of the enamel lobes; even when the explorer does not stick". Rich (18) also is convinced that in order to save the occlusal surfaces of the first permanent molars it is best to place some plastic material in these grooves "though they are not carious or even faulty". Dwyer and Peterson (19) advocated prophylactic odontotomy as soon as pits and fissures are discovered. They add that no pain will be felt as "so little preparation is necessary at that time and that the resulting filling will be durable". It would appear from the latter statement that correct cavity preparation is not an absolute prerequisite toward the attainment of operative success. To this point of view I am unable to accede. Boyd (20) insisted, correctly, that amalgam should never be placed in preparations which are not complete and which do not permit the entrance of the amalgam plugger. He emphasizes that without proper condensation satisfactory results cannot be obtained.

TABLE III

Incidence of caries and defective fissures in the first permanent molars of a group of 111 children between the ages of 5 and 6 years

These pupils were not included in the group of 5246 pupils forming the main substance of this paper.

Number of pupils examined	111	
Number of first permanent molars erupted	167	
Number of carious first permanent molars	2	(1.2%)
Number of non-carious first permanent molars examined	165	
Number of defective fissures disclosed	4	(2.4%)

Hyatt (5) gave interesting tables showing the location of caries, in relation to surfaces involved, in a group of 1,000 industrial workers, male and female, ranging in age from 16 to 25 years. The occlusal surface of the first molar was most often attacked. Because of this finding he urged the cutting out and filling of non-carious first permanent molars as the logical course for the prevention of caries of these teeth. He further explained that there is no need for undercuts, and that cavity preparation is simple for these non-carious organs. In his words, "all that is necessary is to have a flat bottom and smooth walls". He also maintained that when upon examination a sharp and pointed explorer enters a groove and sticks, a fissure is present. When the explorer fails to stick, but the groove appears deep and narrow, "this place must be protected with a prophylactic filling." Hyatt explained that "this is done by thoroughly cleaning out the groove, drying and filling with copper cement or copper amalgam".

Difficulties and disillusions are bound to follow should permanency of a filling be expected where correct cavity preparation has not been employed. In numerous instances young children and even adults are not aware that such "prophylactic" fillings have dropped out long before they are due for reexamination. An opportunity is thus given for food to collect and pack into these cavities.

Fermentation is established, dissolution of enamel by lactic acid takes place, and penetration of caries into the dentin is furthered. Instead of being protected, these teeth are rendered a prey to caries for, as Hyatt maintains, the dentin at this early stage of life is decidedly more susceptible to rapid destruction.

In an examination of 12,753 persons whose ages ranged from 16 to 60 years, Hyatt and Dwyer (21) found that more than 52% of all carious cavities occurred on the occlusal surfaces of the premolars and molars. While at first sight this finding may appear striking, one must consider that a preponderance of cavities on these surfaces does not furnish evidence that they actually developed from preexisting pits or fissures. It may also be noted that the 52% of the teeth showing caries on their occlusal surfaces included not only the first permanent molars but also the first and second premolars and the second and third permanent molars. On the strength of the evidence presented by Hyatt and Dwyer the assumption that "98 per cent of the first permanent molars do become carious" breaks down at its most vital point.

The failure to employ the rubber-dam when filling teeth is vehemently denounced by Prime (22), who maintained that such an omission is bound to result in failure. Yet the advice is tendered that fissures and "etchings" in the permanent teeth be filled immediately upon their eruption. At the early age of 6 years, the application of the rubber-dam is hardly possible. The question arises: Do not the advocates of prophylactic odontotomy stimulate the development of caries by placing fillings "bound to result in failure" in teeth which are not, but may become, carious?

Furniss (23) quoted Gray, of the Leeds Dental Service of England, who wrote in one of his reports that "on the basis that fissures of all teeth will eventually decay, except for a very limited number of children who appear to be immune to dental caries, it is sound policy to remove the fissures at what can be termed the pre-carious stage and to replace them with a metallic filling giving a self-cleansing surface to the tooth". Like many others, Gray fails to weigh the possible complications following the insertion of fillings. In an investigation conducted by Ottolengui (24) on amalgam fillings in extracted teeth, he was able to detect with the aid of a magnifying glass that a fair percentage of these fillings showed recurrent decay around their margins on the occlusal surfaces of the premolars and molars. In a study of extracted teeth carried out by Marshall (25) it was disclosed that 1,746 (32%) of the 5,435 teeth presenting caries, showed secondary decay under and around the margins of fillings. Among the factors responsible for such an occurrence he cites the perplexities encountered in filling operations. These difficulties, I find, are many times increased when working on children.

McLean (26) correctly pointed out that because of their lack of stable chemical and physical properties, certain materials used for filling teeth do not serve the purpose of prevention for which they were intended. Speaking of amalgam fillings he remarks that "caries will be found under many of them". Discussing the silicates, he points out that they disintegrate at the gingival margin where the acidity of saliva is most concentrated and caries ravages the tooth under

the fillings. This is very true; in my clinical studies as well as in my field observations I have repeatedly encountered recurrent decay under and around the borders of many cement, silicate and amalgam fillings.

Bossert, (27) whose studies on the relation of the shape of the occlusal surfaces of molars to the incidence of caries are important, reported that fissures favor the retention of food debris and bacteria, and that they are "usually" carious. He does not, however, support his contention with statistics. There is no more reason to believe that fissures are "usually" carious than there is to suppose that they are the result of causes similar to those resulting in the promotion of caries.

It was Johnson's (28) conviction that only in "extreme cases and in very susceptible mouths" is the filling of a fissure a desirable undertaking. When dealing with children showing immunity, he was never been able to bring himself "to put a drill in an otherwise sound tooth and open a fissure". The fact that many of these teeth never decay leads to the conclusion that it is a pure assumption to believe that they will ultimately become carious (29). McBeath (30) warned against the opening of all fissures in the permanent and deciduous teeth and stated that " . . . indications for such operative intervention must depend upon the needs of the patient". Townend, (31) as a result of his own studies and observations, concluded that if an individual is naturally immune, the fissure, even if defective, will not develop into caries. Bibby (32) maintained that those fissures which have not have been invaded by caries during the first 5 years of tooth life rarely decay, even when defectively developed. Sim Wallace (33) stated that "with regard to so called fissures, say in the molars, these are as a rule normal, but even when they may be abnormal, they may remain free from decay. Hicks (34) pointed out that pits should never be cut as a matter of routine; their conversion into cavities and subsequent filling is not warranted unless there is perceptible evidence that the dentin beneath is actually softened. Howe (35) stated that, in his opinion, "the connection of fissures to the carious process is uncertain." Kronfeld (36) emphasized that "there is no justification for the common assumption that fissures are defects or faulty formation". He further concluded that fissures occur normally on perfectly formed premolars and molars. They are declared to have existed in phehistoric men and are also present in both primitive and civilized races today. It is true that modern man presents a high incidence of caries in these fissures but Kronfeld maintained that this does not furnish proof that fissures are poorly formed, that their enamel is inadequately calcified, and that they are, therefore, predisposed to caries. Often, the inference is made that teeth with fissures are poorly formed, their enamel inadequately calcified and, therefore, they are predisposed to caries. However, despite the often repeated assumption that 99 per cent of all fissures will eventually become carious, I still question whether this is the case. Unfortunately, all the important details on the subject have not been fully considered.

Bodecker (37) implied that during the process of mastication food is forced into the depths of fissures, causing an acid to form there, and that this is responsible for the quick progress of caries in teeth presenting fissures. He further

infers that the apparently different anatomical construction accounts for the lower incidence of caries and the slower progress of decay on the proximal surfaces. His theories here referred to, cannot be considered without serious reservations by the clinical investigator. In many mouths the occlusal surfaces of the premolars and molars remain free from caries even when defective fissures are present. On the other hand, the smooth surfaces of the anterior teeth, or even of the posterior teeth, are markedly attacked by decay. The incidence of caries in such teeth is considerable, as suggested by Prime (22), Townend (38) and others. The presence of a predominantly greater number of cavities on the proximal surfaces of the deciduous teeth (molars included) has been reported by McCall (17) who related that "fully half of the caries found in deciduous molars originates in proximal surfaces". My own investigations revealed a preponderance of proximal over occlusal cavities in close to 500,000 deciduous molars. This subject will be fully discussed in a future report.

In an investigation of 146 university students whose ages ranged from 16 to 21 years, a total of 101 first permanent molars free from caries was found. It is not unlikely that this number had been reduced by prophylactic odontotomy. Three hundred and fifty-eight first permanent molars had been filled. It is stated on good authority that the period of greatest dental decay is between the tenth and sixteenth years of life, therefore these 101 molars may continue to remain non-carious for the rest of the lives of the individuals in whom they were noted. The statement is made that only 1 out of every 2,500 fissures, or about 1 in every 1,000 molars escapes dental caries, yet in this series one out of every 6 molars remained immune (39).

Saunders (40), in 1837 took exception to the belief that indentations of the crowns of molars have any bearing on the cause of caries: first, because caries also attacks smooth surfaces such as the anterior surfaces of the incisors, and molars, and secondly, it is unbelievable "that the Omniscient Architect of the human frame, while succeeding to unbounded admiration in the construction and contrivance of other equally important and more delicate organs should have failed in these and that the teeth alone should require a modification of their form by art". He adds: "The fallacy of a theory which will not apply to one half the phenomena for the explanation of which it is propounded is apparent". Had Saunders' views received the consideration they deserved, we should perhaps today—a century later—be nearer to a solution of the problem of dental caries. Preventive measures would also have been of a different character to those so strongly advocated by the followers of prophylactic odontotomy.

Clinical studies and observations over a period of 21 years, supplemented by follow-up investigations, have convinced me that it is erroneous to assume that all cavities on the occlusal surfaces of premolars and molars have developed from pre-existing fissures, even when these fissures were defective. The examination of nearly 200,000 pupils and observations of over 2 million occlusal surfaces have afforded me an opportunity to learn what I believe is the truth of the matter. The follow-up of a considerable number of children whose occlusal fissures were not opened proved the incorrectness of the statement that most of

the teeth with such fissures will eventually decay. I also learned that a substantial number of first permanent molars without abnormal fissures never decay.

DATA

A study was made of a group of 5,246 pupils (4, 536 white; 710 negro) of Newark, New Jersey. Their ages ranged from 5 to 15 years. Various nationalities and socio-economic levels were represented. A total of 20,004 first permanent molars was considered. The mouth mirror, explorers, college pliers, cotton pellets, and scalers were used in the procedures. The X-ray was not utilized.

According to Hyatt no one can prove that cavities involving more than one surface of this tooth did not have their beginning on its occlusal aspect. Because of this belief all decayed first permanent molars in the present study are registered as "carious" irrespective of the surface on which the cavity occurred. Filled first permanent molars were not taken into account in establishing the extent of caries incidence. Some probably were carious, but others may have been filled in an effort to "prevent" their decay. It seems clear that a true picture of the incidence of this disease cannot be supplied by adjudging all filled teeth as having been previously carious. First permanent molars were typed as carious if at the time of examination they showed signs of decay characterized by the presence of a localized cavity containing disintegrated tooth structure. Previously extracted first permanent molars were included as carious. Those with poorly coalesced fissures but without actual cavitation were not considered carious, but were grouped separately.

Of the total number of 14,750 first permanent molars studied in the white boys and girls (Table I) (filled teeth and those with defective fissures were not included), 5,275, (35%) were carious. The negro boys and girls disclosed a lower percentage: a fraction over 26% of the 2,610 first permanent molars investigated were found affected (filled teeth and those with defective fissures were not included). The influence responsible for a lesser susceptibility to caries of these teeth in the negro could not be determined. Dietary factors, individual habits of oral hygiene, and social status were investigated but, as yet, have failed to furnish a definite answer.

The negro girls showed a smaller percentage of carious first permanent molars than the colored boys. The percentage of carious molars in the white boys and girls was nearly identical. The relation of sex to caries incidence in these teeth while suggestive in the colored, appeared of no consequence in the white children. Further studies in this direction are necessary. If the white children are considered separately, it will be noted that those in the 13 to 15 age groups revealed the highest percentage of carious first permanent molars. This was also the case with the negro boys and girls comprising the same age groups (13 to 15 years).

Impressive as these findings may be, let us examine the evidence more closely. We must first of all realize that the evidence does not confirm the statement that 98 per cent of these teeth do become carious. Eight and 9 years have elapsed

since their eruption, yet considerable numbers have resisted the inroads of decay. The conviction expressed by Bibby (32) gains support, namely, that those fissures which have not been invaded by caries during the first 5 years of life rarely decay, even if they are defective. My studies agree with this conclusion. (Table IV).

In no way is it inferred that all cavities in the older groups developed from pre-existing defective fissures. A great part of the increased number of carious first permanent molars in these groups may be due to the advent of prophylactic odontotomy. Obviously, with an increased number of filled non-carious first permanent molars, the percentage of those free from caries was equally reduced.

A point not to be ignored is that the majority, although not all, of the molars affected showed occlusal caries. Proximal and compound cavities were displayed by a number of them. That the compound cavities did not originate on the proximal aspect of the teeth is most difficult to prove. How can one conceive that compound cavities involving only the very marginal ridge or end of the

TABLE IV

Incidence of caries, freedom from caries, defective fissures and filled first permanent molars in a group of 576 white and negro children between the ages of 13 and 16 years

These children were not included in the 5246 pupils who form the main substance of my study.

	WHITE		NEGRO		TOTAL
	Boys	Girls	Boys	Girls	
No. of pupils studied	146	145	142	143	576
No. of molars free from caries	177	180	294	294	945
No. of molars with defective fissures	26	25	36	59	146
No. of molars filled	141	202	38	39	420
No. of carious molars	155	102	156	128	541
No. of missing molars (extracted because of caries)	85	71	44	52	252

occlusal aspect of these teeth had their beginning in an occlusal pit or fissure? I do not deny that caries may and does develop in normally formed occlusal surfaces of first permanent molars, as a number of investigators convincingly assert. However, the wisdom of cutting out perfectly formed enamel in teeth which are not, but possibly may become, carious does not appear scientifically justified. Here the fundamental error of prophylactic odontotomy asserts itself.

When the additional data is considered it is found that of the 4,536 white pupils studied, 1,105 showed 4 first permanent molars each free from caries (598 boys and 507 girls). Of the total of 710 negro children investigated, 309 (163 boys and 146 girls) showed 4 first permanent molars each free from caries. These facts are significant, for they point out the extent of immunity to caries of these teeth in many individuals.

In both sexes and races, the lower left first molar ranks first in susceptibility; the lower right molar is next in order. The immunity of the first molars in the

upper arch is considerably above that of those in the lower—12% fewer upper carious molars in the girls and 11% fewer upper carious molars in the boys were noted in the white children and a similarly substantial decline in the number of upper first permanent molars so affected in the negro boys and girls. (Table II).

Table IV gives a statistical history of findings in an additional group of 576 children investigated. Their ages ranged from 13 to 16 years. A total of 2,304 first permanent molars were considered. Filled teeth were grouped separately. Indeed it would be a mistake to conclude that all have been previously carious. Those with defective fissures were also grouped separately. It is significant that the negro children with the lowest number of filled molars and with the largest number of fissures revealed the largest number of such teeth free from caries. One important observation is that a substantial number of defective fissures failed to develop into caries after a lapse of 6 or more years, a finding supporting Bibby's idea. The socio-economic status of the children comprising this group differed considerably. The white children belonged to families of good financial circumstances; they could afford not only reparative work but also such "preventive" measures as prophylactic odontotomy and, as a matter of fact, there seemed reason to suppose that such measures had been taken. The negro children, on the contrary, were members of very poor families and it seems quite reasonable to deduce from the findings accumulated that in their case prophylactic odontotomy was seldom if ever performed. This factor is to be considered, as it seems to explain in some measure the reason for the lower percentage of caries-free molars in the white children.

SUMMARY

From clinical studies and field observations as well as from statistical studies of the incidence of caries in the first permanent molars of 5,933 children it appears erroneous to assume that "99 out of every 100 fissures will have to be filled some day" (Hyatt), and that "98 per cent of the first permanent molars do become carious" (Bodecker), and that 95 per cent of these teeth are defective on eruption (9). The evidence revealed in these studies does not point in that direction.

REFERENCES

1. Dunning, W. B., and Davenport, E. J., *A Dictionary of Dental Science and Art* (Philadelphia, Blakiston: 1936) p. 215.
2. Hyatt, T. P., Prophylactic Odontotomy the Ideal Procedure in Dentistry for Children, *D. Cosmos*, **78**: 353, 1936.
3. Bodecker, C. F., Bacteria in Relation to Enamel Fissures, *D. Items of Interest*, **48**: 797, 1926.
4. *Idem.* The Tooth Brush in Relation to Occlusal Fissures, *D. Items of Interest*, **48**: 161, 1926.
5. Hyatt, T. P., The Conservation of Non-carious First Permanent Molars, *J. A. D. A.*, **13**: 655, 1926.
6. Munblatt, M. A., Preventive, Therapeutic and Reconstructive Dentistry, *D. Digest*, **36**: 88, 1930.
7. Black, A. D., Prevention in Dentistry, *J. A. D. A.*, **13**: 582, 1926.
8. Prime, J. M., Controlling Dental Caries, *J. A. D. A. & D. Cosmos*, **24**: 1950, 1937.
9. Harris, S. D., Why Fill Newly Erupted Teeth? *D. Survey*, May 1934.

10. Brekhus, P. J., A Report on Dental Caries in 10,445 Students, 1928–1930, *J. D. Res.*, **11:** 487, 1931.
11. McFall, W., Practical Prevention in Pedodontia, *J. A. D. A. & D. Cosmos*, **25:** 1017, 1938.
12. Starr, E. E., Significance of Early Dental Caries, *J. A. D. A.*, **26:** 1089, 1939.
13. Rich, C., Prophylactic Care of the Child's Mouth, *J. A. D. A.*, **22:** 1592, 1935.
14. McCall, J. O., The Modern Program for Prevention of Dental Disease, *J. Canad. D. A.*, **4:** 615, 1938.
15. Cotton, W. A., Report of a Study of the Diet Factor in Relation to Dental Decay in Children and the Most Probable Food Offender, *Nutrition & D. Health*, March 1937.
16. Anderson, B. G., Clinical Study of Arresting Dental Caries, *J. D. Res.*, **17:** 443, 1938.
17. McCall, J. O., Lessons to be Learned in a Children's Dental Clinic, *J. A. D. A.*, **21:** 1869, 1934.
18. Rich, C., Discussion to Dr. Hyatt's paper on: The Conservation of Non-Carious First Permanent Molars, *J. A. D. A.*, **13:** 661, 1926.
19. Dwyer, H. S., and Peterson, T. D., Review of Progress in Children's Dentistry, *J. 2nd Dist. D. Soc. (N. Y.)*, **24:** 179, 1938.
20. Boyd, D., Modern Operative and Preventive Procedures for Children, *Rev. Den. Child.*, **6:** 6, Third Quarter, 1939.
21. Hyatt, T. P., and Dwyer, H. S., A New Aspect of Prophylactic Odontotomy, *J. A. D. A.*, **18:** 1356, 1931.
22. Prime, J. M., Inconsistencies in Operative Dentistry, *J. A. D. A. & D. Cosmos*, **24:** 82, 1937.
23. Furniss, A., Notes on the School Dental Service, *D. Mag. & Oral Topics*, **55:** 1072, 1938.
24. Ottolengui, R., The Failures with Amalgam as Commonly Used, *D. Cosmos*, **67:** 999, 1925.
25. Marshall, A. J., Dental Disease versus Dental Therapeutics, *J. South. Cal. D. A.*, **4:** 1462, 1937.
26. McLean, D. W., Tooth Decay—the Universal Disease, Chapter IV, These Teeth of Mine, *Hygeia*, **13:** 326, 1935.
27. Bossert, W. A., Tooth Form—Cause of Caries, *D. Survey*, Nov., 1934.
28. Johnson, C. N., Preventive Dentistry; its Scope and Possibilities, *J. A. D. A.*, **17:** 346, 1930.
29. *Idem*, The Control of Dental Caries, *D. Items of Interest*, **55:** 161, 1933.
30. McBeath, E. C., Comments on Dentistry for Children in General Practice, *D. Outlook*, **26:** 218, 1939.
31. Townend, B. R., personal communication.
32. Bibby, B. G., personal communication.
33. Wallace, J. S., personal communication.
34. Hicks, P. B., Mouth Examination, *D. Students Mag.*, Nov. 1937.
35. Howe, P. R., personal communication.
36. Kronfeld, R., First Permanent Molar; Its Condition at Birth and its Postnatal Development, *J. A. D. A.*, **22:** 1131, 1935.
37. Bodecker, C. F., Cause of Rapid Destruction of Occlusal Enamel Fissures, *D. Items of Interest*, **53:** 21, 1931.
38. Townend, B. R., *Dental Inspection and Treatment, Thirtieth Annual Report of the School Medical Officer* (County Council of the West Riding of Yorkshire: 1937) p. 12.
39. Brucker, W. H., The Incidence of Involvements of the First Permanent Molar. A Statistical Investigation on the Findings in Six Hundred First Permanent Molars in One Hundred and Five College Freshman, Thesis, (Phila., Thomas W. Evans Museum and Dental Institute: March 1941.)
40. Saunders, E., *Advice on the Care of the Teeth*, (London, Thomas Ward: 1837), p. 58.

STUDIES ON THE INCIDENCE AND CAUSE OF DENTAL DEFECTS IN CHILDREN[1]

VII. Fissures and Caries

MARCU BRUCKER, D.D.S.

Dental Division, Bureau of Health Education and Service, Board of Education, Newark, N. J.

Many investigators have devoted deserved attention to the study of the relationship of fissures to susceptibility to dental caries. Different opinions have been expressed as to whether or not fissures are defects. A capital stand was taken by Kronfeld (1) who considered them "normal". Broomel (2), Friesell (3), and others qualified fissures as "structural defects", while Hyatt (4), Bodecker (5) and Breckhus (6) classified them as "faults" in the enamel. Howe (7) regarded fissures as "the space between abutting enamel coverings". According to Dunning and Davenport (8) a fissure may be defined as a "developmental linear fault. . . commonly the result of the imperfect fusion of the adjoining dental lobes".

Various statements have been made as to the incidence of defective fissures on the occlusal surfaces of the first permanent molars. For example, it is emphatically stated that "surface cracks, or fissures, are present in fully 85% of all molars coming through the gums" (9).

DATA

My studies fail to support this contention (Table II). One author (10) does not hesitate to consider fissures as affecting 95% of these teeth. I find no confirmation of this theory, for in my study of a group of 5,001 pupils, with a total of 11,410 non-carious first permanent molars, a totally different picture was revealed. The ages in this group ranged from 5 to 15 years and both sexes were included. Various nationalities and socio-economic strata were represented. There were 670 negroes and 4331 white children in the group.

In my investigation molars were considered non-carious if, at the time of examination, they failed to reveal a history of decay. Those which had been filled were not included in this study, for their previous history in this era of prophylactic odontotomy was difficult to ascertain. At the risk of repetition it must be stated that my analysis is limited to a presentation of facts as disclosed by a meticulous examination of 11,410 first permanent molars free from caries and on which no operative work had ever been performed. I adhered strictly to the policy of registering only those fissures which showed imperfect coalescence of the enamel lobes. The explorer, mouth mirror, college pliers and cotton pellets, and also the scalers were used in the investigation. The x-ray was not employed.

[1] Received for publication March 17, 1943. Revised by author June 10, 1943.

The investigation disclosed a total of 1,296 defective fissures in the 9,463 first permanent molars considered in the white children, and a total of 147 imperfect fissures in the 1,947 first permanent molars studied in the negro children. The incidence of faulty fissures was lower in the white boys than in the white girls, but in the negro boys and girls the percentage of defective fissures was quite similar (Table I). The influence of sex and race appears mixed and confusing; still, the difference in the incidence of this defect in the white and colored children is interesting. It was noted that in the white children the lower right first permanent molar was most frequently affected. In the negro children the

TABLE I

Incidence of defective fissures in the 4 first permanent molars of boys and girls

	BOYS				GIRLS			
	Teeth							
	$\underline{6}\vert$	$\vert\underline{6}$	$\overline{6}\vert$	$\vert\overline{6}$	$\underline{6}\vert$	$\vert\underline{6}$	$\overline{6}\vert$	$\vert\overline{6}$
No. of molars studied:								
White	1364	1381	1041	1113	1244	1327	1002	991
Negro	*267*	*280*	*246*	*251*	*240*	*249*	*214*	*200*
No. of molars with fissures:								
White	139	170	176	161	157	156	189	148
Negro	*13*	*15*	*21*	*29*	*9*	*15*	*23*	*22*
% with fissures:								
White	10.1	12.3	16.8	14.3	12.5	11.7	18.7	16.1
Negro	*4.8*	*5.3*	*8.8*	*11.5*	*3.7*	*5.9*	*10.5*	*10.6*

TABLE II

Incidence of caries and defective fissures in the first permanent molars of a group of 203 children between the ages of 5 and 6 years

These children were not included in the group of 5001 pupils forming the main substance of this paper.

Number of pupils examined	203	
Number of first permanent molars erupted	316	
Number of carious first permanent molars	9	(2%)
Number of non-carious first permanent molars examined	307	
Number of defective fissures found	9	(3%)

lower left first permanent molar revealed the highest incidence of this defect. In both races and sexes, the upper molars presented a substantially lower incidence of defective fissures.

DISCUSSION

Advocates of prophylactic odontotomy insist that 98% of the occlusal surfaces of the first permanent molars become carious. They also insist that the factor most responsible for this condition is the presence of imperfect fissures on these aspects of so many of these teeth. I find it impossible to attach any weight to

this argument. To my knowledge, neither of these claims has ever been substantiated by clinical proof, and my findings lead me to believe that they never can be.

SUMMARY

The data here presented indicate that caries of the first permanent molars cannot be directly related to the presence of defective fissures. The disproportion between the incidence of caries and that of faulty coalescence of the enamel lobes is so tremendous that a relationship between the two is out of the question.

REFERENCES

1. Kronfeld, R., First Permanent Molar: Its Condition at Birth and its Post Natal Development, *J. A. D. A.*, **22**: 1131, 1935.
2. Broomel, I. N., personal communication.
3. Friesell, H. E., personal communication.
4. Hyatt, T. P., A Statistical Study of the Location of Dental Caries Shows the Practical Value of Prophylactic Odontotomy, *D. Digest*, **34**: 235, 1928.
5. Bodecker, C. F., Procedure for Fissure Eradication, *D. Items of Interest*, Quart. Rev., **5**: 45, 1930.
6. Brekhus, F. J., A Report on Dental Caries in 10,445 University Students (1928–1930), *J. A. D. A.*, **18**: 1350, 1931.
7. Howe, P. R., personal communication.
8. Dunning, W. B., and Davenport, E. J., *A Dictionary of Dental Science and Art* (Philadelphia, Blakiston: 1936), p. 215.
9. Metropolitan Life Insurance Company, *Good Teeth* (New York), p. 5.
10. Harris, S. D., Why Fill Newly Erupted Teeth?, *D. Survey*, May 1934.

A STUDY OF THE BILATERAL INCIDENCE OF CARIOUS LESIONS[1]

DAVID B. SCOTT, D.D.S.

From the Division of Dental Research, University of Rochester School of Medicine and Dentistry, Rochester, N. Y., and the Baltimore College of Dental Surgery, Dental School, University of Maryland, Baltimore, Md.

Statistical studies of dental caries have indicated that lesions tend to occur in equal or near-equal numbers in both sides of the jaws. Leigh (1), Tylman (2), Hyatt (3), Driak (4), Bodecker (5), Day and Sedwick (6), Knutson, Klein and Palmer (7), Van Huysen and Moon (8), Cheyne and Drain (9), Brekhus (10), Healey and Cheyne (11), and others (12) have provided evidence which demonstrates the remarkable uniformity in which carious lesions occur in the left and right teeth.

Bilateral caries is the production of destructive lesions in identical surfaces of corresponding teeth situated on opposite sides of the mouth. *Unilateral* caries refers to a pair of similarly located surfaces, one of which is carious, the other caries-free. In a consideration of either condition, it is necessary to examine the teeth in pairs, left and right, in the same mouth. A third state in which a pair of corresponding teeth may be bilaterally affected in respect to one pair of surfaces and unilaterally in respect to another is referred to in this study as *mixed* caries.

Former studies of dental caries (1, 2, 4, 11) paid little attention to destruction of bilaterally identical surfaces in the same individual. References were made to the fact that approximately the same number of teeth on the right side were affected as on the left but no evidence was provided that caries-susceptibility is equal for the two sides of the mouth. The methods employed made it possible that unilaterally affected teeth in some persons were compared with corresponding teeth from the opposite side in others. Such procedures would result in an apparent bilaterally equal distribution of caries which may not be an accurate indication of true bilateral affection.

It is difficult to present statistical data regarding bilateral caries because considerably more accurate means of observation and registration of paired teeth are required than seem to be practical for large scale in vivo studies. Results obtained from the usual observations of decayed teeth, most of which are performed without radiographic examination, may be noticeably altered by use of radiographs (13). This is true especially in cases where bilateral proximal caries is proceeding at different rates, and evidence of attack may be detected by the oculo-instrumental method on one side, whereas the incipient breakdown of the corresponding surfaces on the other side may be visible only on the radiograph.

In the present study attention is given to the incidence of bilateral lesions in

[1] Received for publication, November 22, 1943. Revised by author Jan. 31, 1944.

the posterior teeth as observed radiographically. The limitations of radiographic diagnosis of caries are recognized and taken into account in evaluating the data.

METHOD

Three hundred individual pairs of left and right posterior bite-wing (interproximal) radiographs from the files of the Department of Oral Roentgenology of the Baltimore College of Dental Surgery are the basis of this study. No radiographs showing bone destruction or periodontal involvement were included for observation. Each pair was required to show permanent teeth only, and to present clear views of the maxillary and mandibular premolars, and first and second molars, when they were present. No attempt was made to select radiographs of persons in any age range. Carious and restored dental surfaces and missing teeth were recorded. Observations from 2400 pairs of bilaterally situated teeth (16 teeth in each pair of films) were tabulated.

It was assumed that a restoration represents an attack by caries, and if there is no evidence of periodontosis, missing teeth are absent by virtue of caries. According to Bodecker (5), each missing tooth corresponds to a mean of 3 cavities. Determination of the caries type was accomplished by comparison of bilaterally identical surfaces of corresponding left and right teeth of the same jaw in the same individual. The condition of a lesion, filling, or missing tooth on one side occurring with a lesion, filling, or missing tooth opposite was classified as bilateral caries. If a lesion, filling, or missing tooth occurred opposite a normal (caries-filling-free) surface, the condition was classified as unilateral. Bilateral and unilateral conditions occurring simultaneously in corresponding left and right teeth are referred to as mixed caries.

FINDINGS

A synopsis of the findings is indicated in Tables I and II. Without regard to pairs, 60.2% of the total number (4500) of posterior teeth visualized in the radiographs were carious. The incidence of lesions was almost equal in each of the quadrants of the mouth, slightly higher in left than in right dentition, and greater in maxillary than in mandibular teeth, observations confirmed by other investigators (10, 11).

Three hundred teeth were missing: in bilaterally identical pairs, 142; unilaterally, opposite a carious or filled correspondent, 126; and opposite a normal correspondent, 32.

Consideration in left and right identical pairs of the 9000 interproximal surfaces observed in the radiographs revealed bilateral lesions due to caries in 171 pairs, to restorations in 247 pairs, and to a lesion on one side and a filling on the other in 126 pairs. Sixty-four intact interproximal surfaces were without opposite correspondents by virtue of missing teeth. Further unilateral disease was observed in 350 pairs as a result of carious lesions, and in 297 as a result of dental restorations. Interproximal surfaces bilaterally free from caries or restorations numbered 6490, more than two-thirds of the total. Four hundred

seventy-three pairs (40%) of bilaterally identical molar teeth, contained occlusal restorations bilaterally. Fifty-eight pairs (5%) of premolar teeth were likewise restored.

Of the posterior teeth 73.1% decayed, missing, or filled were involved bilaterally. Fifteen percent of these were unilaterally carious on another surface. The remaining 26.9% of the posterior teeth were affected unilaterally only.

TABLE I

Distribution of caries in 4800 posterior teeth as indicated in bite-wing radiographs

	NUMBER OF TEETH	PERCENT		
		Of teeth decayed (2,709)	Of teeth studied (4,800)	Of teeth visualized (4,500)
Carious				
Bilaterally only	1,574	58.1		
Mixed involvement	406	15.0		
Total bilaterally	1,980	73.1		
Unilaterally only	729	26.9		
Total carious	2,709	100.0	56.4	60.2
Caries-free	1,791		37.3	39.8
Missing	300		6.3	
	4,800		100.0	100.0

TABLE II

Distribution of caries in the posterior teeth of 300 persons as indicated in interproximal radiographs

TYPE OF CARIES	NUMBER OF PERSONS	PERCENT
Bilateral only	114	38.0
Bilateral, unilateral and mixed	172	57.3
Total bilateral	286	95.3
Unilateral only	6	2.0
Caries-free	8	2.7
	300	100.0

Bilateral caries was found in 95.3% of the individuals represented in the radiographs. Only 2% revealed a condition of pure unilateral lesions; and less than 3% were free from evidence of caries. The carious members of unilaterally carious left and right tooth pairs were almost uniformly distributed in the oral quadrants, left and maxillary teeth being slightly in the majority. Unilaterally carious teeth of the maxillary left quadrant numbered 155; of the maxillary right, 140; of the mandibular left, 158; and of the mandibular right, 123.

DISCUSSION

The possible presence of occlusal, buccal, or lingual caries not apparent in radiographic images of teeth classified as caries free is recognized. The effect of such lesions in these teeth would be to increase the total caries incidence and the number of tooth surfaces bilaterally and unilaterally affected. Any figures expressing or dependent on the incidence of lesions found in the radiographs observed in this study may be considered as minimal.

The order of incidence of bilateral carious lesions in individual right and left pairs of teeth was similar to the order in which lesions were observed in the teeth without regard to pairs, (*fig. 1*). The mandibular first molar tooth appeared to be affected more frequently than any other posterior tooth. The mandibular

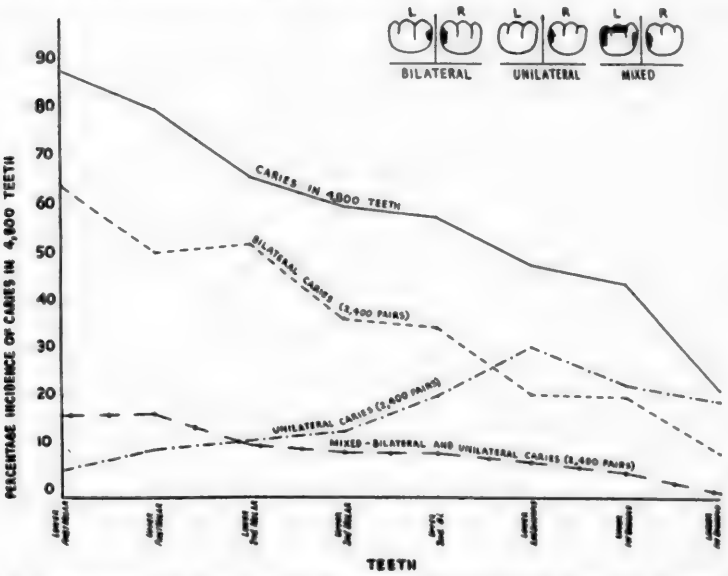


Fig. 1. Distribution of gross, bilateral, unilateral and mixed caries in posterior teeth individually and in pairs.

first premolar tooth was least often carious. Unilateral lesions tended to occur in a reversed order as compared with that of bilateral caries. The mandibular second premolar tooth apparently was attacked more often by unilateral caries than other posterior teeth. Mixed lesions were distributed similarly to bilateral lesions, the first molar teeth apparently being most frequently involved in bilateral and unilateral lesions occurring simultaneously.

The differences in the numbers of unilaterally diseased teeth situated in the various quadrants are not significant and indicate that caries probably does not begin more readily in one side than in the other, or in one jaw more than in the other.

The findings indicate a marked tendency toward bilateral symmetry in car-

ious lesions of the posterior teeth. The comparatively low incidence of unilateral lesions may possibly be explained by a difference in the status of development of lesions required to complete symmetry. It is impossible to state that unilateral lesions may later become bilateral, but the distribution of cavities by quadrants indicates that either member of a left and right tooth pair may become carious first.

Since it was desirable to obtain a conception of the incidence of caries without regard to age, this factor was not considered in the investigation. The results represent a static rather than a dynamic picture of dental caries from which certain present and prognostic deductions may be made.

Etiology. The possibility of bilateral caries is suggested in the anatomy (3, 14), arrangement and development of the teeth. Their bilaterally symmetrical form and alignment and paired eruption appear to render equally susceptible the teeth of each side. Further, anatomic and pathologic structural variations may be expected to occur bilaterally and thereby act as local influencing factors. In posterior teeth most caries is encountered in grooves, pits, and fissures, which occur symmetrically, a fact constituting convincing evidence of the significance of anatomic configurations in the production of bilateral lesions. In the present study, relatively immune surfaces such as the mesial surfaces of the first premolar teeth were found bilaterally intact in nearly every instance, while relatively susceptible surfaces such as the wide flat mesial aspects of the maxillary first molar teeth demonstrated almost universal bilateral involvement.

Clinical Significance. The knowledge that a tendency toward a bilateral occurrence of carious lesions exists is important from the standpoint of diagnosis and prognosis of the disease. Observation that a specific surface is carious or restored suggests examination of the corresponding surface of the same tooth of the opposite side. This procedure may be valuable as a method for disclosing obscure cavities and is a well founded aid in rendering a complete diagnosis. Here, bite-wing radiographs have proven themselves highly satisfactory. Considering the mouth as an entity, if there is a marked tendency toward bilateral caries it is not unreasonable to assume that pairs of teeth unilaterally carious will subsequently become bilaterally involved.

SUMMARY

A study of the posterior teeth of 300 individuals as visualized in bite-wing radiographs revealed that almost three-fourths of the teeth involved with lesions were affected symmetrically in bilaterally identical pairs. The tendency toward a bilateral incidence of caries was supported by consideration of etiologic and predisposing factors generally conceded to be active in caries production. Knowledge of the tendency is important in the diagnosis and prognosis of carious destruction of the teeth.

REFERENCES TO LITERATURE

1. Leigh, R. W., *Military D. J.* **6:** 183, 1923.
2. Tylman, S. D., *J. A. D. A.* **14:** 1379, 1927.

3. Hyatt, T. P., *D. Digest* **34:** 235, 1928.
4. Driak, F., *Zeitsch. Stomatologie* **27:** 743, 1929.
5. Bodecker, C. F., and Bodecker, H. W. C., *D. Cosmos* **73:** 707, 1931.
6. Day, C. D. M., and Sedwick, H. J., *D. Cosmos* **77:** 442, 1935.
7. Knutson, J. W., Klein, H., and Palmer, C. E., *J. A. D. A.* **25:** 1923, 1928.
8. Van Huysen, G., Moon, H. S., and Davidson, S., *J. D. Res.* **18:** 261, 1939.
9. Cheyne, V. D., and Drain, C. L., *J. D. Res.* **19:** 571, 1940.
10. Brekhus, P. J., *J. A. D. A.* **28:** 1126, 1941.
11. Healey, H. J., and Cheyne, V. D., *J. A. D. A.* **30:** 692, 1943.
12. Bertram, F. P., and Brown, J. E., *J. A. D. A.* **30:** 1392, 1943.
13. Burket, L. W., *J. D. Res.* **20:** 71, 1941.
14. American Dental Association Committee on Research in Dental Caries. *Dental Caries*, 2nd ed. (New York: Lancaster Press: 1941), p. 244.

A CRITICAL MICROSCOPIC TECHNIC APPLIED TO STUDY OF HUMAN DENTIN[1]

WARDLAW M. HAMMOND,[2] LESTER W. BURKET, D.D.S., M.D.[3] AND SEYMOUR J. KRESHOVER, D.D.S., PH.D.[4]

From the Dental School, University of Pennsylvania, Phila., Pa. and the Medical School, Yale University, New Haven, Conn.

The investigation of ordinary objects which are well within the resolving power of the microscope may be conducted without attention to refinements in technic. In the examination of structures which approach the limits of vision, illumination becomes quite as important a factor as the quality of the lenses in the optical train and the thinness of the preparations studied.

True resolutions at high magnifications necessitate a wide cone of light and the use of a full or close-to-full numerical aperature (N. A.) of the objective. The angle of light thrown upon the objective should never be in excess of that required to just fill the aperture. For searching and for preliminary observations, further cutting down of the light cone can be resorted to in order to prevent eye strain. When the subject is found under these latter conditions the aperture must then be increased until the cone of light just fills the objective. Provided that the optical train of the equipment is in true alignment, and provided that the illumination is absolutely axially correct a true resolution will be obtained.

In the examination of delicate structures such as thin unstained sections of decalcified dentin, where the darkest portion is but little different from the background, even the slightest haze or glare will destroy the image. If these conditions and others are not met, spurious optical effects are easily produced when examining structures of fine detail which approach the limits of vision, such as the dentinal fibrils and tubuli.

Some of the photomicrographs in the current text books of dental histology do not appear to meet the above conditions.[5] Consequently, the structural arrangement of these tissues as interpreted from these false resolutions may be incorrect. An example of this is shown in *figs. 1* and *2* which are photomicrographs of preparations of unstained, teased dentinal fibrils as viewed with diffuse and direct lighting. The corona areas are present on both photomicrographs but they are most prominent in the reproduction made with direct

[1] Read at the 20th General Meeting of the International Association for Dental Research, New York City, March 14–15, 1942. (*J. D. Res.* **21**: 299, 1942.) Received for publication, Feb. 6, 1944.

[2] Research Associate in Microscopy, Dental School, Univ. of Penn.

[3] Assistant Professor Oral Medicine, Dental School, Univ. of Penn.

[4] Pathology Department, Medical School, Yale University.

[5] Churchill, H. R., Translation and Revision of Meyer's *Normal Histology and Histogenesis of the Human Teeth and Associated Parts.* (Philadelphia, Lippincott: 1935), p. 64.

lighting. Meyer stated that with direct illumination artificial Neumann's sheaths are produced as the result of interference and diffraction phenomena. While this conclusion is in keeping with the resolutions he illustrated, the latter would not be considered true resolutions by critical microscopists. He ignored the fact that a full or near-to-full N. A. of the objective in use is necessary for a true resolution when the magnification is of a high order. With the highest power object glasses the limit of resolution is reached and it is with these lenses that refinements of illumination become of the utmost importance.

Meyer suggested that the photomicrograph taken with diffuse lighting represented the actual structure of the dentinal fibrils. This is a misleading statement. His photomicrograph (*fig. 1a*), using diffuse lighting, was taken with an extremely narrow cone of illumination which prevented it from being a true resolution. This is proved by the corona which is observed surrounding the fibrils. While direct lighting at high magnification is more difficult to handle than diffuse lighting, it gives much better results when properly used. We have studied teased, unstained dentinal fibrils and thin sections of both stained and unstained human dentin. In the study of the teased, unstained dentinal fibrils we have found that results similar to those depicted by Meyer in *fig. 1a* could be produced only when a narrow cone of light was used for illumination, i.e., it is not a true resolution which can be obtained only with a full or near-to-full N. A. of the objective.

A resolution of teased, unstained dentinal fibrils in which a wide light cone and a near-to-full N. A. were used is shown in *fig. 3*. The teased fibres appear to have real structure and the over and under crossing of the two fibres can be clearly seen. In examining such a teased preparation or the thin decalcified sections under high magnification, it is evident that several planes of undulating fibres or tubuli are present. It is necessary to select only one for a resolution. The best method is to select a fibre or tubule in the upper plane of the specimen and to give it a wide cone of illumination, as much as it will stand without glare or flare. A better appreciation of the structural character of these unstained, teased dentinal fibrils can be obtained with the use of dimensional lighting (*fig. 4*). This clearly illustrates the rounded nature of the teased dentinal fibrils.

Accurate resolutions of extremely thin ground and decalcified sections of human dentin were also studied utilizing the requisites for accurate resolution which have been enumerated previously. With sufficient care to technical details satisfactory resolutions may be obtained on thin, unstained ground sections (*fig. 5*). A resolution made from an unstained, decalcified section of human dentin also shows a tubule, an open space, and a tubular wall which appears structurally different from the dentin matrix (*fig. 6*). The tissues forming the internal boundary of the dentinal tubules have a different density from the dentinal matrix. *Fig. 7* is a resolution of a thin, unstained, decalcified section of human dentin which was taken with a transmission band of 370 to 510 mμ and a near-to-full numerical aperture. Dimensional lighting was not used for this resolution. The structural differences of the inner boundary of the dentinal tubules are readily apparent.

These observations made on unstained, ground and decalcified sections of human dentin preclude the possibility that in stained preparations the deeper staining of the boundary of the dentinal tubules is an artefact. The staining of

FIG. 70a.—Isolated dentinal fibrils. Photomicrograph obtained with the aid of diffuse light. Unstained. Magn. ± 1000x.

1A

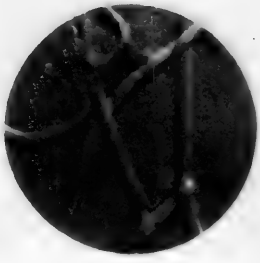
FIG. 70b.—Dentinal fibrils of Fig. 70a. Photomicrograph obtained with the aid of direct light. Artificial sheaths of Neumann are produced as the results of interference—and diffraction phenomena.

1B

FIG. 68.—Isolated dentinal fibrils, unstained. Magn. ± 1000x.

2

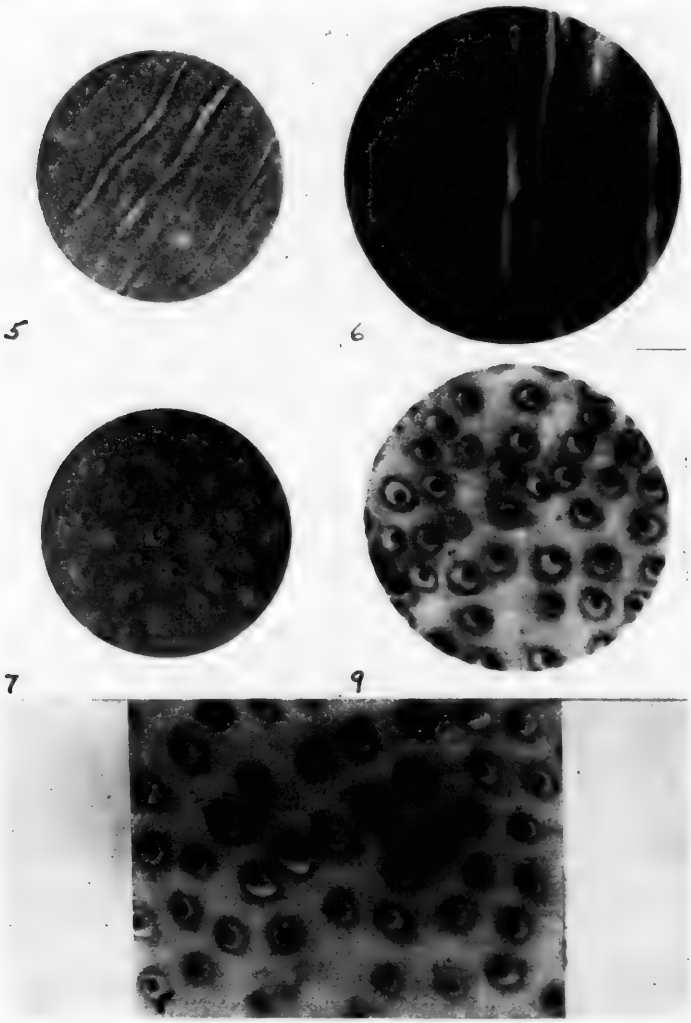
3



4

Figs. 1 to 4

biologic tissues is a selective phenomenon depending on the affinity of the particular cells, or cellular products, for the various components of the stains. It is not a mass coloring or dyeing effect.



Figs. 5 to 9

Differences in appearance between the dentin matrix and the boundaries of the dentinal tubules are slightly accentuated by light staining of the decalcified sections of human dentin (*fig. 8*). The crescentic shaped areas surrounding the fibrils in the dentinal tubules in this and other photomicrographs are correctly seen only when wide light cones and a near-to-full N. A. of the objective are used. We have not attempted to interpret these areas other than to assert that

they are not artefacts resulting from the photomicrographic technic and we do not believe they are artefacts in the histologic technic. The examination of lightly stained, decalcified cross-sections of human dentin revealed a fine structure-channel or fibres, directly connecting the walls of the dentinal tubules, which are difficult to record photographically (*fig. 9*). Facilities for accurately measuring these fine channels or fibres were not available, but assuming the dentinal tubules to be 1.3 to 2.2. micra in width these connecting channels are approximately 100 to 200 A° or 200 10^{-7} mm. in depth or diameter. These fine channels or structures appear microscopically to have a definite connection between the walls of the tubules which leads us to believe that they may have a specific function. These structures should not be confused with the branching of the dentinal fibrils.

In one of the decalcified cross-sectional preparations of stained human dentin, two clear-cut dentinal fibrils were observed within one tubule (*fig. 9*). The examination of longitudinal sections of dentin could not reconcile this finding as a point of branching of the dentinal fibril as at such points of branching each fibre has its own tubule.

SUMMARY

Some of the essential requirements for obtaining accurate resolutions and photomicrographs of minute objects which approach the limits of vision have been discussed. Resolutions failing to meet these essential requirements not only serve as illustrations in modern text books but they have been used as objective proof for certain theories and hypotheses concerning the structure of the dentin. Failure to meet these essential requirements is shown by the corona effects in the photomicrographs and the stated method of illumination which was used for their production.

Our resolutions of thin sections of stained and unstained, decalcified and ground sections of human dentin reveal structural differences from those commonly illustrated. The selection of our material, the photomicrographic and the histologic technics employed lead us to believe that the microscopy of the human dentin, and perhaps other dental tissues, should be reconsidered using accurate and refined technical methods. No attempt has been made to interpret the structures depicted.

A STUDY OF THE PATTERN AND COMBINATIONS OF CONGENITALLY MISSING TEETH IN MAN[1, 2]

PETER J. BREKHUS, CLARENCE P. OLIVER AND GEORGE MONTELIUS

University of Minnesota, Minneapolis, Minn.

According to the established dental formula, man has 32 teeth in his permanent set. It is frequently observed that many persons fail to develop 1 or more of their third molars, thereby causing the formula to lie between 28 and 32. A small percentage of persons also fails to develop even that allotted number. A tooth that has failed to develop is considered to be congenitally missing.

The congenital deficiency in teeth cannot always be considered a detrimental anomaly provided there is no associated glandular defect which is more harmful in its reaction. Actually, the deficiency may represent a prediction of man's future dental formula. However, the deficiency is a disturbing factor for some persons, particularly if the absence of the teeth mars the facial features. Some affected persons are extremely conscious of their anomalies, especially if the anterior teeth are involved.

Several theories have been offered to explain the congenital absence of teeth. One finds rather frequently in the literature the suggestion that the dental anomaly is associated with congenital syphilis. Although congenital syphilis can and does cause anomalies of the teeth, many persons who lack teeth or have abnormally shaped teeth are not syphilitic, and syphilitic stigmata and dental anomalies should be considered as separate entities.

Congenitally missing teeth are considered by some investigators to be the expression of an evolutionary trend. A reduced dentition would thus be related to the attempt by nature to fit the shortening of the arches. In support of the theory, Schultz (11) cited the high frequencies with which the third molars in the posterior part of the arches and the upper second incisors and lower first incisors in the anterior portion are missing. However, he did not consider the second premolars which are also missing very frequently and yet do not have a like anatomical relationship to the ends of the arches. Those investigators who suggest that the theory of disuse may satisfactorily explain the congenital absence of teeth in modern man will also find it difficult to explain the absence of second premolars. It is hard to establish a relationship with function or disuse which can explain why the first premolar, the second molar, or possibly the first

[1] This study was made possible by financial aid from the Committee on Human Heredity of the National Research Council and by supplemental aid from the Graduate School, the Dight Institute and the Dental School of the University of Minnesota.

[2] Read at the 21st General Meeting of the International Association for Dental Research, March 13–14, 1943, Chicago, Ill. (*J. D. Res.* **22**: 197–198, 1943). Received for publication January 31, 1944.

molar, is preserved at the expense of the second premolar. In rare instances, moreover, the canines are the only teeth congenitally missing, and they are anatomically the best developed teeth in man.

Montagu has developed the hypothesis that anomalies of the upper second incisors are linked to abnormal development in the fusion of the maxillary and premaxillary bones (9). That theory fits in well with the belief held by some workers that anomalies of upper second incisors are closely related to cleft palate. Developmental anomalies at the fusion point of the two bones might lead to defects of the second incisors. It is also conceivable that anomalies in the development of the mandibular symphysis might result in anomalies of the lower first incisors. Although defects in the bone development may cause dental anomalies, it is not necessarily true that all of the anomalies of the incisors are due to the developmental anomalies which cause the bone defects. Deficiencies in incisor teeth seem to be part of a more general condition expressed by deficiencies in other teeth as well, including the second premolars which are not associated with fusion points of the bones.

The belief that endocrine dysfunctions may be associated with dental defects is often quoted. Some of the more extreme cases of missing teeth are definitely due to endocrine disturbances. Thadani has reported on a total absence of teeth in males (14). Other workers have found anodontia or extreme partial anodontia in females as well as in males. The affected individuals also tend to have other conditions suggestive of ectodermal dysplasia. Wheelon reported 4 cases of dental defects of the upper second incisors, some of them having family histories, in patients who had clinical symptoms of other endocrine involvements (17). He referred to a report by Kaplan who believed that upper second incisor deficiencies are related to gonadal deficiency. Obviously the large number of offspring produced by some persons who lack upper second incisors shows that extreme gonadal deficiency is not a factor.

Downs and Cohen and Anderson have reported that mentally defective persons are more likely to have congenitally missing teeth than are mentally normal persons. Downs reported in his study that approximately 50% of the mentally defective persons lacked some teeth as compared to approximately 17% of the normal persons (5). Cohen and Anderson found that 10.6% of mental patients and only 2.5% of a group of normal persons lacked upper second incisors. They also reported a higher incidence of missing lower first incisors in the mentally deficient individuals (3). Downs concluded, however, that dental anomalies could not be considered pathognomonic of any definite type of endocrine dysfunction.

Some investigators suggest that heredity is a factor in causing the congenital absence of teeth. Although heredity may be a factor, the general tendency to revert to heredity as an explanation of conditions difficult to interpret otherwise leads often to a misinterpretation of the actual value of heredity. Primarily heredity is used to explain the transmission, incidence, and variability of a trait. The method of inheritance does not in itself explain the origin of an anomaly. The genes which are inherited act in some manner to control a developmental or physiological process, and if the resulting development differs from normal the

variation will be expressed as an anomaly. The defect in the body may also be caused by an endocrine disturbance that is inherited. Different genes may control dissimilar developmental processes and yet be expressed in like manner in the finished product, that is, the anomalies of the body. Montagu recently pointed out the possibility that upper second incisor anomalies might be due to 2 distinct developmental defects, those related to "dental genes" and those associated with "maxillary genes" that lead to abnormal fusion of maxillary and premaxillary bones (9).

MATERIALS AND METHODS

Over a period of 2 years (1941–43), 11,487 patients of the Dental Clinic at the University of Minnesota were examined. Every patient who had a dental anomaly was subjected to more complete examination. A total of 202 persons, including 16 who came to the clinic because of relationship to patients with dental deficiencies, were investigated fully. Seven of the cases for various reasons can not be used in the study. The other 195 cases of dental anomalies are used in this report.

Although an attempt was made to record every dental anomaly (particularly congenitally missing teeth) in the patients, 2 possible discrepancies should be noted in calculating population frequencies. Firstly, it was not possible for 1 operator to inspect all the patients of the clinic. With more than 1 person making the examinations, the human factor makes it possible that some cases were missed. The 195 cases, therefore, only approximate the actual number of dental anomalies in the 11,487 patients. Secondly, the sex factor must also be considered. Sixty percent of our cases of tooth deficiencies were observed in females. We do not have the ratio of the sexes in the patients of the clinic but the greater proportion of them were females. If the sexes differ in the incidence of the dental anomalies, as has been indicated by Hrdlicka (6) for upper second incisors, that fact will be expressed in our data. The nationalities of the patients were mixed. Most of them belonged to the Caucasian race. Practically all of the cases which were collected through the clinic were of adults or children who should have developed all their permanent teeth, with the possible exception of the third molars. The average age of the 195 persons was 22.9 with the median age 21.

Complete intraoral radiographs, as well as complete mouth impressions for the purpose of making models, were made of each person with dental anomalies. In selected cases, photographs were taken of the individuals. At the time impressions and radiographs were made, data on the familial histories were also collected. All interpretations are based upon examinations of the patients, followed by a study of the dental models and radiographs. In some instances, personal histories were necessary in order to ascertain whether the teeth had been extracted.

DATA

The 195 recorded cases of dental anomalies include 5 with enamel deficiency (in 3 families), one with opalescent dentin, and one with hypoplastic enamel.

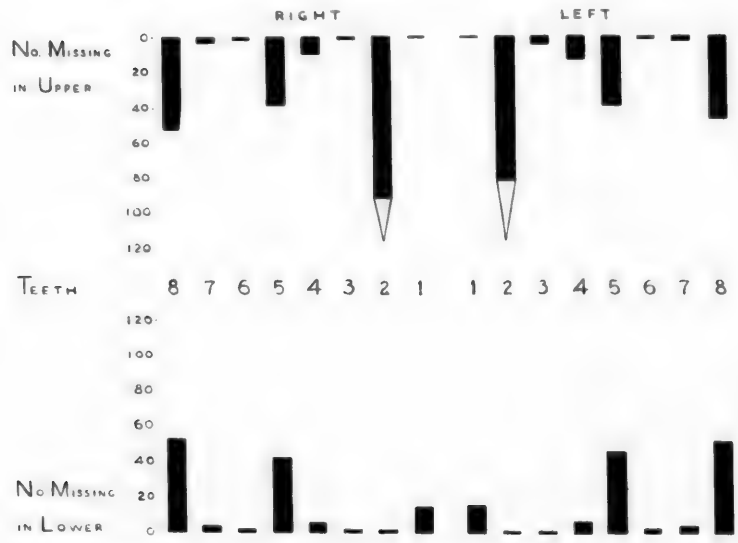


Fig. 1. Observed pattern of missing teeth. Graphic representation of pattern of missing and peg-shaped teeth observed in 184 persons. For the upper second incisors, the solid portion represents the number of missing teeth, the peaked portion the added number of peg-shaped teeth. The total number of missing teeth was 644.

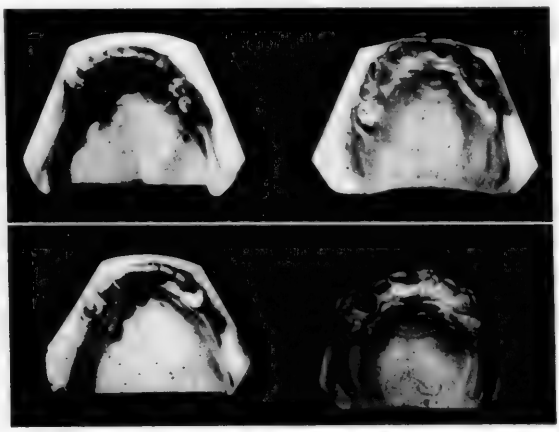


Fig. 2. Arches of mother and daughter congenitally lacking posterior teeth. Upper casts are those of a woman 47 years old who lacked her molars and two second premolars. The lower casts are her daughter's who lacked her molars and four second premolars. (Illustrations from *Your Teeth*, by Peter J. Brekhus [Minneapolis: University of Minnesota Press, 1941]. They are reproduced with the permission of the publisher).

(One other case of hypoplastic enamel was observed in a person who also had congenitally missing teeth and was therefore counted in the study.) In four of the cases the only anomaly was the presence of supernumerary teeth. However,

supernumeraries were also observed in a few persons with congenitally missing teeth. For obvious reasons the above 11 cases were not included in the material on congenitally missing teeth. The other 184 cases represent individuals who lacked 1 or more of their teeth (congenitally missing), or had abnormally shaped teeth (pegs); a few cases had noticeably small (pigmy) but not malformed teeth.

In 7 of the 184 cases, extreme deficiencies were observed in which from 14 to 19 teeth were congenitally missing. Two of the 7 extreme cases occurred in members of one family. Photographs of the arches are shown in *fig. 2*. A mother lacked all her molars and the 2 left second premolars (*fig. 4*). One of her daughters had no molars or second premolars. A second daughter and 2 sisters of the mother had complete sets of teeth. The mother knew of no other individual in the kinship who lacked any teeth. It is of interest that the extreme dental deficiencies in the mother and daughter have not marred their facial

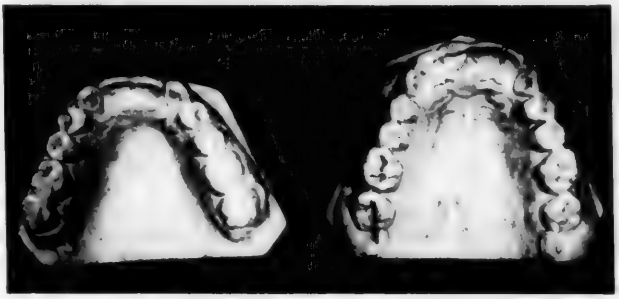


FIG. 3. Photographs of small arch with $\frac{2|2}{}$ deficient but normal alignment, and of normal arch. Cast on left shows an arch lacking upper second incisors with no spaces retained between first incisors and canines. That on right shows an upper arch normal in size and in number of anterior teeth; third molar can be observed on left side.

features; the arches have grown normally even though the posterior teeth failed to develop.

Severe dental deficiencies are recognized symptoms of some diseases. Complete or partial anodontia, for example, is associated with the sex-linked anhidrotic ectodermal dysplasia and with the recessive, incompletely sex-linked epidermolysis bullosa. With the possible exception of the mother and daughter referred to (*fig. 4*), the extreme cases of dental deficiencies in our records are probably the result of endocrine dysfunction or other diseased conditions. One patient had the dental formula:

$$\frac{7\ 6\ \text{III}\ 1/1\ 4\ \text{V}\ 6\ 7}{7\ 6\ 3\ /\ 2\ 3\ \text{IV}\ \text{V}\ 6\ 7}.$$

This patient, a boy of 17 years when brought to the clinic, was very conscious of the deficiency and had become timid because of the criticisms of his playmates. Prosthetic work gave the boy a presentable appearance and a new outlook on life. He was the only affected member in a sibship of 6 persons, although,

according to the information gathered from his mother, the boy's father lacked his upper second incisors. The probability that the severe dental deficiency in the boy was due to a more general diseased condition is suggested by his medical history.

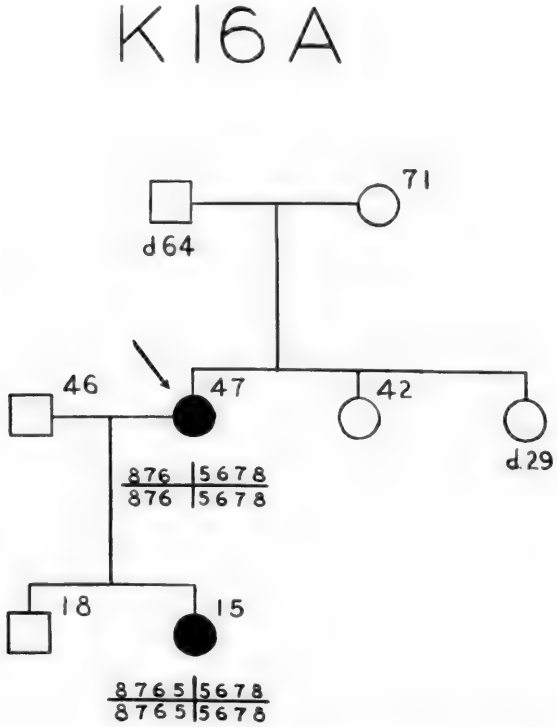


FIG. 4. Family history with severe condition of congenitally missing teeth. Numerals used as superscripts show ages of the persons; d indicates that person is dead and numeral represents age at death. Dental formulae represent teeth which are missing in persons with solid circles. Propositus is shown by arrow. Squares represent males; circles, females. Photographs of arches of 2 affected persons are shown in *fig. 2*.

PATTERN OF MISSING TEETH

Overwhelming evidence indicates that any one of the 32 teeth may occasionally be congenitally missing without being associated with glandular disturbance or other diseased conditions. However, some teeth are more likely to be missing than are others.

In our 184 cases, 644 teeth were congenitally missing and 59 were peg-shaped. The data are shown in Table I and represented graphically in *fig. 1*. Upper second incisors were most frequently deficient. The right upper second incisor was missing in 92 instances and peg-shaped in 25. In the left maxillary arch, the upper second incisor was lacking in 82 cases and peg-shaped in 34. Next in

frequency were the third molars and the second premolars in all 4 quadrants. Lower first incisors were absent with equal frequency on the right and left sides of the mandibular arch. Although they were missing less frequently than were second premolars, the lower first incisors were absent more often than were the maxillary first incisors, mandibular second incisors, and the canines, first and second molars in both arches.

No significant differences were observed between the right and left halves of either arch. The only significant differences between the maxillary and mandibular arches were observed in the anterior teeth. First incisors in the mandibular arches were lacking more often than were the first incisors in the maxillary arch. Lower second incisors were rarely missing but the upper second incisors were missing more frequently than any other tooth.

An explanation for the variations in frequency with which the different teeth are missing is difficult to find. The pattern shown in *fig. 1* is the same as that found in our first group of 100 cases studied, and is in general agreement with

TABLE I

The number of missing and peg-shaped teeth observed in 184 persons

	RIGHT								LEFT							
No. missing in upper..	53	3	2	39	10	2	25/92	1	1	34/82	5	13	39	2	3	47
Teeth.....	8	7	6	5	4	3	2	1	1	2	3	4	5	6	7	8
No. missing in lower..	52	3	2	42	5	2	2	15	16	1	1	6	46	2	3	52

For the upper second incisors, the upper numbers represent the "pegs"; the lower, the missing teeth.

the reports by Bengstrom (1), Dolder (4) and Werther and Rothenberg (16). Most of our observed congenitally missing canines, first and second molars, upper first incisors, and lower second incisors were found in the so-called extreme cases. The 2 cases of congenitally missing first molars were found in the mother and daughter shown in *fig. 2* and *fig. 4*. Their condition fits that generally found, that an absence of first molars is associated with an absence of other molars, and differs from the condition reported by McCall and Samuels (8) in which 4 cases of missing first molars had other molars present. Some of the other teeth which are rarely missing may be absent even in the less extreme cases. Although the canine teeth are rarely missing, a few individuals in our records, including some who do not belong to the extreme cases, lacked one or more of the cuspid teeth. Dolder and also Werther and Rothenberg have reported cases of missing canines. Montagu found that 3 out of 528 persons lacked canines. There have also been reports by Schweitzer, Lubner and Thompson of persons in whom only canine teeth were missing (15). A case has come into our records since this report was prepared in which a person lacked only his 4 permanent canine teeth.

OBSERVED COMBINATIONS OF MISSING TEETH

Missing and malformed teeth were studied in 184 persons for whom both models and radiographs were made. Of those, 178 cases involved the more frequently missing teeth (upper second incisors, second premolars, third molars) individually or in combinations. Some combinations also included other teeth. Six cases involved only those teeth that are missing relatively infrequently. Included among the 6 were 4 cases with only the lower first incisors missing, 1 case with only an upper left first premolar absent, and one with a lower right canine missing. The individual with the canine absent also had hypoplasia in the lower first incisors and lower first molars.

TABLE II

Observed combinations of missing teeth in 184 persons

DENTAL COMBINATIONS	NUMBER OF CASES				
	USI	2 Pm	3M	Misc.	Totals
Only one kind	60	22	4	6	92
USI, Misc.	6			6	6
2Pm, Misc.		5		5	5
3M, Misc.			4	4	4
USI, 2Pm.	7	7			7
USI, 2Pm, Misc.	3	3		3	3
USI, 3M.	30		30		30
USI, 3M, Misc.	3		3	3	3
2Pm, 3M.		15	15		15
2Pm, 3M, Misc.*		4	4	4	4
USI, 2Pm, 3M.	6	6	6		6
USI, 2Pm, 3M, Misc.**	9	9	9	9	9
Total cases	124	71	75	40	184

USI—upper second incisor; 2Pm—second premolar; 3M—third molar; Misc.—any other teeth. "Only one kind" indicates that only one type of tooth was absent.

* Includes two extreme cases.

** Includes five extreme cases.

The data on the 184 cases are shown in Table II. The vertical columns show the types of teeth involved, the first horizontal column shows the number of cases which had respectively only upper second incisors, second premolars, third molars or miscellaneous teeth missing. All the other horizontal columns represent the various combinations of missing teeth as designated. All kinds of teeth other than the upper second incisors, second premolars, or third molars are grouped into a single category designated as miscellaneous. One hundred and twenty-four of the 184 cases involved at least the upper second incisors. Seventy-one lacked 1 or more second premolars. Seventy-five were lacking 1 or more third molars. Forty had anomalies in the miscellaneous group of teeth.

We find that exactly 50 % of the 184 cases lacked more than one kind of tooth (Table II). Among the 124 persons who had anomalies of the upper second

incisors, 51.6% were also deficient for other kinds of teeth. Of the 71 cases lacking second premolars, 69% failed to develop other kinds of teeth as well. The 75 observed cases of missing third molars included 94.7% that also lacked some other kind of tooth. Among the 40 cases with miscellaneous teeth missing, 85% also lacked either the upper second incisors, second premolars, third molars, or a combination of these teeth. It is recognized, however, that the observed number of persons lacking third molars does not represent the true population frequency. Since the third molars are absent so frequently, persons are likely to come into the clinic only if they have some other difficulty with their teeth. It can only be stated that the reported number represents those cases of missing third molars observed in the patients, or their relatives, with other congenitally missing teeth.

POPULATION FREQUENCIES

The determination of the true incidence of congenitally missing teeth must be based upon the primary contact persons or index cases, referred to as propositi.

TABLE III

Incidence of combinations of missing teeth based upon 168 propositi

COMBINATIONS OF ABNORMAL TEETH	NUMBER OF CASES	OBSERVED INCIDENCE IN %	EXPECTED INCIDENCE IN %	RATIO: OBS.–EXP.
USI, 2Pm	23	0.200	0.00555	36–1
USI, 3M	46	0.400	0.00588	68–1
2Pm, 3M	30	0.261	0.00350	75–1

USI—upper second incisor; 2Pm—second premolar; 3M—third molar.

There were 168 propositi with congenitally missing teeth. The relative proportions of propositi with only one kind of missing tooth and with the various combinations do not differ significantly from those reported for all 184 cases.

Of the 168 propositi, 111 were deficient in upper second incisors, 66 in second premolars, 70 in third molars and 37 in other (miscellaneous) teeth. The incidence of dental deficiencies among all patients (11,487), as measured by the propositi, is therefore 1.5%. The incidence of missing and abnormal upper second incisors among the patients of the clinic is 0.966%; of missing second premolars, 0.575%; of missing third molars, 0.609%; and of missing miscellaneous teeth, 0.322%.

Combinations of missing upper second incisors, second premolars and third molars in the same individuals occurred more often than should be expected purely as a matter of chance. Among the 168 propositi, 23 had abnormal upper second incisors as well as second premolars, 46 had both upper second incisors and third molars involved, and 30 had abnormal second premolars and third molars.

The observed incidences for the combinations of missing teeth in the 168 persons are presented in column three of Table III. In the fourth column are

shown the expected frequencies of combinations as based upon the observed frequencies for each kind of tooth. The last column represents the ratio of the observed to the expected incidences. In each combination, the observed frequency far exceeds that expected as a matter of chance.

It is obvious that mere coincidence cannot explain the high incidence of combinations. The explanation of the frequent association of one type of missing tooth with another type is not easy to determine from our data. It is possible that a person inherits a tendency which is general in nature. As a consequence, an individual would have an inherent developmental defect related to tooth development which in interaction with one or more extraneous or inherent factor might be expressed in one of several ways including multiple deficiencies in the same person.

The tendency for more than one kind of tooth to be deficient is observed among the members of some families. In family K132A (*fig. 5*), the propositus, the ninth member in generation III, lacked

$$\frac{8\,5\,2\,/\,2\,5\,8}{8\quad 1/\,1\quad 8}.$$

Her mother lacked all third molars; a sister lacked two third molars. A brother had all his teeth although one canine was impacted. Four brothers and sisters developed all their teeth. One niece, a daughter of the brother with the impacted canine, lacked a second premolar. Another niece whose parents had complete sets of teeth lacked an upper second incisor and a second premolar. Three nieces had no missing teeth.

In family K108A (*fig. 6*), the propositus, member 5 of generation II, lacked her upper second incisors and a canine. One of her nine siblings lacked upper second incisors. One of the children of the propositus had anomalies of the upper second incisors, a canine and three third molars, and another lacked second premolars and the lower third molars.

Relatives of the propositus in K11A also show variation in the dental anomalies (*fig. 7*). The father of the propositus failed to develop one canine. The propositus, the fourth member in generation II, had anomalies only of the upper second incisors. One of her siblings had peg-shaped upper second incisors and missing third molars. Another had one peg-shaped upper second incisor, missing maxillary canines, and missing third molars. A third sibling lacked only canines. One sibling had a complete set of teeth.

It should be stated, however, that multiple deficiencies in persons who lacked upper second incisors were found approximately as frequently in persons who had no family histories of deficiencies as they were in persons who had affected relatives.

It is possible that the high incidence of multiple deficiencies in our cases may indicate that the cases observed in the clinic do not represent a random sample of the general population. Unless some pathological condition caused the patient to come to the dental clinic, a person with multiple deficiencies is more likely to seek dental aid than is one who lacks only one or one kind of a tooth.

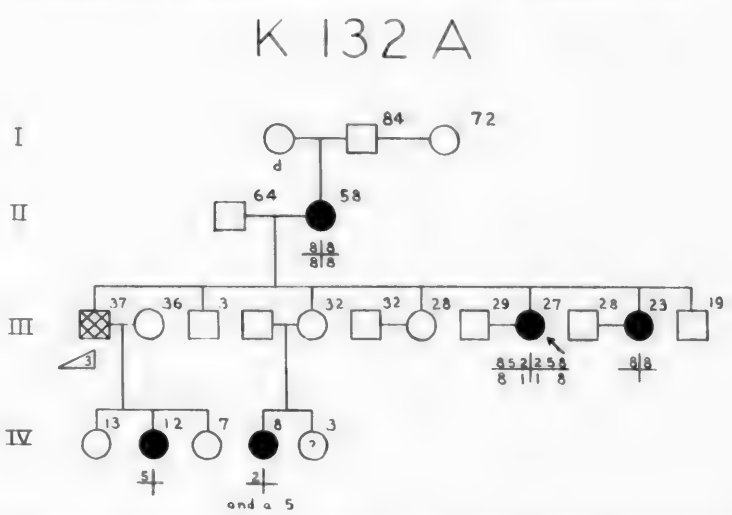


FIG. 5. Kinds of dental anomalies observed in one family. See *fig. 4* for explanations. ◁ indicates an impacted upper right canine. ? indicates that data are not certain. Each sibling and mother of propositus have been contacted.

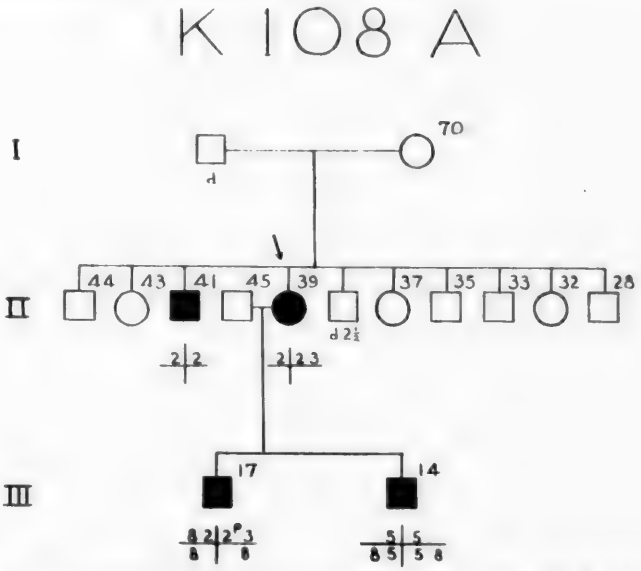


FIG. 6. Pedigree chart of family with congenitally missing teeth. See *fig. 4* for explanations. Radiographic examinations were made of propositus and of the 2 members of generation III.

Some persons are unaware that they lack teeth. The lack of observance of the deficiency may sometimes be due to the closing of the spaces or to the retention of the deciduous teeth in that area. The fact that several instructors made the

examinations may also help to explain the high incidence of multiple deficiencies. Individuals may lack congenitally the second premolars, but the appearance will suggest to some observers that the condition is due to extractions rather than to congenital absence. Unless those persons are questioned, the cases will be missed.

That the data may not represent a random sample is supported by the low incidence of congenitally missing teeth observed in the patients of the clinic. The incidence observed is 1.5. In comparison, Bengstrom reported that of 563 dental radiographs he studied, 6.5% showed evidence of congenital lack of teeth. The high frequency observed in his study is due in part at least to the type of cases he studied; they were orthodontia cases. Dolder found 3.4% of the 10,000

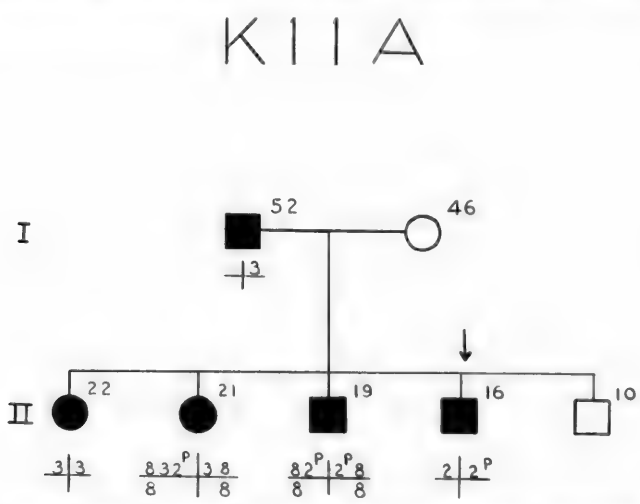


FIG. 7. Family history showing variable expressions of dental anomalies. See *fig. 4* for legend.

children that he studied had dental deficiencies, and Werther and Rothenberg reported 2.3% of 1,000 children affected. Approximately 1% of our propositi had anomalies of upper second incisors. The incidence is lower than that reported by many other workers. Cohen and Anderson (3) found 2.5% of 1,466 school children deficient in upper second incisors. Campbell (2) found a frequency of 1.75% in 1,000 persons studied. Hrdlicka (6) has reported a frequency of 1.4% for males and 3% for females. Montagu (9) found 5.1% of upper second incisors missing or abnormal in shape. Rose (cited by Schultz, 11) found a variation in frequency depending upon the nationality of the person studied.

It is evident that the incidences determined by the several workers have varied considerably. Our observed incidence is the lowest yet reported. Any study based upon school children, if it includes several grades, is likely to include more

than one member of a family and, therefore, is apt to show a frequency of dental anomalies higher than the true incidence for the population, since dental anomalies do tend to recur in relatives. Consequently, the inclusion of related persons in the data would be expected to show a higher proportion of affected persons than would be found in data based upon non-related persons.

In the compiled population frequencies, 16 of the 184 cases of congenitally missing teeth in our records were eliminated as primary cases (propositi) because the records indicated that they had come to the clinic as a result of our contacts with affected relatives. Only 2 relatives were counted as primary contacts. The incidences found in our study are with the one exception, therefore, based upon persons who are not related.

ARCH SIZE AND CONGENITALLY MISSING TEETH

The suggestion has frequently been made that the loss of teeth is concomitant with a gradual decrease in the size of the jaw in man. Although a deficiency in the number of teeth may cause some decrease in the size of the arches, it is

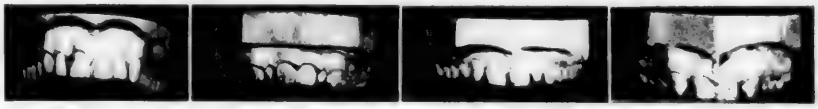


FIG. 8. Three expressions of upper second incisor anomaly. Left cast shows normal set of teeth. In the second cast both second incisors are peg-shaped. In third, right is missing and left peg-shaped. Both upper second incisors are missing in right cast and spaces can be observed between upper first incisors and the canines. (These illustrations are from *Your Teeth*, by Peter J. Brekhus [Minneapolis: University of Minnesota Press, 1941]. They are reproduced with the permission of the publisher.)

difficult to establish a causal relationship between the two conditions. Some persons have arches which are large enough so that spaces are retained where anterior teeth are absent (*fig. 8*). In some persons who lack their anterior teeth, the arches are small and the teeth are evenly aligned with no observable spaces between the teeth (*fig. 3*). Others have too many teeth for the size of the arches, resulting in a crowded condition. In one family in our records, all 3 of the conditions occur. A mother lacks both upper second incisors. Her upper arch is small and the teeth which are present completely fill the arch. Her son has a condition similar to that observed in the mother. Her daughter lacks the second incisors but the spaces have been retained. The mother's sister developed all her teeth in a small arch, and as a consequence, the teeth are crowded. A son of this sister lacks a second premolar, and the teeth which he developed are crowded into a small arch.

In another family, a mother and one of her daughters lack all the molars; the daughter lacks all 4 second premolars and the mother lacks 2 on one side. Yet the arches do not noticeably vary in size from a normal arch (*fig. 2*).

Arch size and tooth deficiency seem to be independent entities. He whose arch is the proper size to fit the number of teeth is a rare individual.

SUMMARY

The pattern observed in a group of persons with congenitally missing teeth is repeated in group after group and suggests that the process is going on in an orderly fashion. The observation may lead to the speculation that the human dentition is gradually readjusting from the original formula of 32 to a lesser number sufficient to satisfy the modern demands.

Individual types of teeth vary in their frequency of absence. As shown by Montagu, the upper second incisors have an anatomical association with the points of articulation of the bones which may be suggestive of a causal relationship. A similar association can be shown for the lower first incisors. However, there is no such anatomical structure relationship with the second premolars that can explain the high incidence with which those teeth are missing. Yet they fit into the general pattern in such a way that one can readily believe that the relationship between deficiencies of the teeth and the articular joints of the bones may be merely coexistent rather than causal, or at best may explain only some part of the cases of congenitally missing teeth. Moreover, the deficiency of the anterior teeth is often observed in more than one member of a family, indicating that the anomaly has not occurred purely as a matter of chance. Whether the frequently missing third molars might be explained on the basis of their relationship to the growth of the arch is uncertain.

Combinations of missing teeth in which more than one kind of tooth is absent are found in many persons. In the cases reported, approximately one-half of the individuals had the multiple deficiencies. Based upon the frequencies with which the individual types of teeth are involved, the combinations occurred too often to be due to chance alone. Not over 7 of the cases of multiple conditions can be explained as due to definite diseases, and those 7 cases lacked from 14 to 19 teeth. The other cases tend to involve only the upper second incisors, second premolars and third molars and not the teeth which are more stable in the arches.

In some families, the dental deficiencies suggest that the inherent defect is not always expressed alike in every affected member of the family. Different kinds of teeth are involved in different members of a family. The probable relationship between peg-shaped and missing teeth will be discussed in another report.

In our data the incidences observed for missing teeth are lower than those reported by some other workers. In part, the differences in frequencies may be explained by the fact that our data include, almost without exception, only one affected member of a family.

Arch size and tooth deficiency seem to coexist but seem to have no causal relationship. Some arches are too small to permit an even alignment of the number of teeth in the arch. In some arches, spaces are retained even though the teeth do not develop. Other arches are small enough that the smaller number of teeth fit the available room and no spaces are observable even though the number of teeth is less than normal. In some persons who lack posterior teeth, the arches have continued to grow, thus leaving space for the teeth which do not

develop. As a consequence, the facial angles in those cases do not differ from the normal expectancy.

REFERENCES TO LITERATURE

1. Bengstrom, R. J., A study of the time of eruption and root development of the permanent teeth between six and thirteen years. Masters Thesis (Northwestern Univ. Dental School, 1930).
2. Campbell, D. K., Congenitally missing upper lateral incisor teeth. *D. Cosmos*, **76**: 459, 1934.
3. Cohen, J. T., and Anderson, J. E., Note on the eruption of the permanent teeth in a group of subnormal children, including an observation of the frequency of congenitally missing laterals. *J. Genetic Psychology*, **39**: 279, 1931.
4. Dolder, Statistical survey of the deficient dentition. (Cited by Thoma, p. 155.)
5. Downs, W. G., Studies in the causes of dental anomalies. *Genetics*, **12**: 570, 1927.
6. Hrdlicka, A., Further studies on tooth morphology. *A. J. Physical Anthrop.*, **4**: 141, 1921.
7. Lubner, B., A case of absence of permanent canines. *Brit. D. J.*, **62**: 262, 1937 (cited by Thoma, p. 192).
8. McCall, J. O., and Samuels, I. M., Congenital absence of first permanent molars. *D. Items of Interest*, **65**: 315, 1943.
9. Montagu, M. F. Ashley, The significance of the variability of the upper lateral incisor teeth in man. *Human Biol.*, **12**: 323, 1940.
10. Oliver, C. P., Brekhus, P. J., and Montelius, G. A., A genetic study of congenitally missing teeth. *Proc. Minn. Acad. Sci.*, **10**: 9, 1942.
11. Schultz, A. H., The hereditary tendency to eliminate the upper lateral incisors. *Human Biol.* **4**: 34, 1932.
12. Schultz, A. H., Inherited reductions in the dentition of man. *Human Biol.*, **6**: 627, 1934.
13. Schweitzer, G., *Deutsche Zahn., Mund. u. Kieferh.*, **1**: 236, 1934 (cited by Thoma, p. 193).
14. Thadani, K. I., A toothless type of man. *J. Heredity*, **12**: 87, 1921.
15. Thoma, K. H., *Oral Pathology* (St. Louis, Mosby Co.; 1941).
16. Werther, R., and Rothenberg, F. *Am. J. Orthodontics*, **25**: 61, 1939 (cited by Thoma, p. 155).
17. Wheelon, H., Clinical significance of the congenital absence of the upper lateral incisor teeth. *Endocrinology*, **9**: 35, 1925.

CONGENITAL PARTIAL HEMIHYPERTROPHY INVOLVING MARKED MALOCCLUSION[1]

CHARLES E. RUDOLPH, D.D.S., AND RUDOLPH W. NORVOLD, D.D.S., M.S.D.

School of Dentistry, University of Minnesota, Minneapolis, Minn.

True hemihypertrophy involves not only the soft tissues of the body but the hard tissues as well. Individuals in whom hypertrophy is so pronounced as to be readily discernible are so rare that up to 1939 (1) only 103 cases had been reported.

Hemihypertrophy is generally described as being of 3 types: Total, in which an entire side of the body is involved; Partial, in which only a portion of the body is involved; and Crossed, in which some part of each side of the body is involved.

The following case is one of a congenital partial hemihypertrophy. Because of its unusual nature and because of the fact that accumulated data may eventually lead to a fuller knowledge as to the etiology of the condition, it was felt that a report of the case would be valuable.

CASE REPORT

S. H., a girl age 9¼ years, was referred to the Graduate Clinic in Orthodontics at the University of Minnesota School of Dentistry in November, 1940, because of evident malocclusion and facial swelling. The left side of her face, especially throughout the maxillary region, presented a swollen appearance (*fig. 1*).

The girl had been a full term baby and weighed 8 lb. 6¼ oz. at birth. Her development apparently was normal. She stood at about 8 months and took her first step at about 10 months. In her early childhood she had a number of diseases, i.e., measles, chicken pox, whooping cough, mumps and scarlet fever with otitis media. Her tonsils and adenoids had been removed at six years. Her general health has been good although she has had occasional colds which are often accompanied by otitis media, especially in the right side. The asymmetry of her face was first noticed at the age of about 2 months. At the age of 3 or 4 years it was very evident (*fig. 4*).

Family History. The patient has 2 brothers both of whom appear to be normal. However, her mother and maternal grandmother have a unilateral facial hypertrophy. The patient's mother has a picture of herself at the age of approximately 8 months in which there is a suggestion of asymmetry and another picture taken at the age of 3 or 4 years which definitely shows the condition (*fig. 3*). In both the mother and the daughter the asymmetry is on the left side of the face. The condition can be observed in the mother today but it is not as pronounced as in the daughter. The mother informed us that her mother also had this asymmetry but to a still lesser degree. Consideration of the grandmother, mother and daugher suggests an inherited anomaly which is becoming progressively more evident.

Physical Examination. A general physical examination by Dr. Charlotte Fisk, in November, 1940, showed the girl to be well within normal limits for her age with the exception of the localized area of hypertrophy. Following are the data: Age: 9¼ years, Height: 55¾

[1] Presented at the 22nd General Meeting of the International Association for Dental Research, Chicago, March 18–19, 1944. Received for publication March 1, 1944.

inches, Weight: 67½ pounds, Nationality: Mother—English and Norwegian; Father—English and Scotch, Flory bone age (as shown by carpal x-rays): 8 years, Cranium: No asymmetry noted, Face: A very definite enlargement on the left side apparently centered in the maxil-

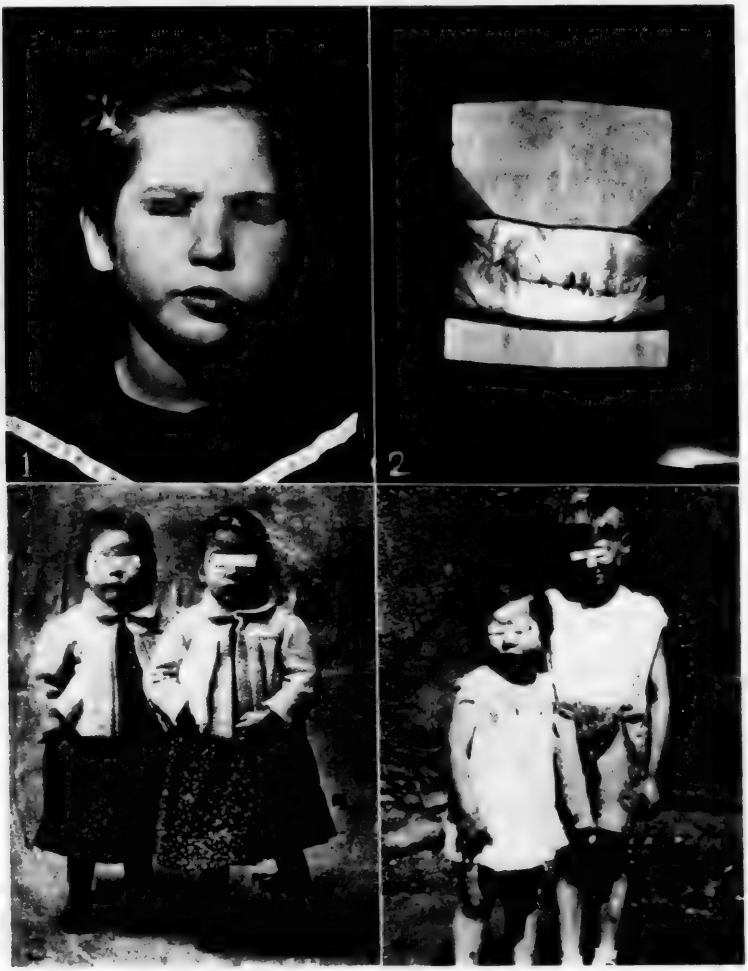


FIG. 1. Patient appearance at 11 years of age. Note swollen appearance of left maxillary area.

FIG. 2. Plaster reproduction of mouth.

FIG. 3. Patient's mother (left) at age of 4 years, showing similar hemihypertrophy.

FIG. 4. Patient at age of 4 years.

lary region. Radiographs shows the hypertrophy to include the bone and teeth as well as the soft tissue. The left antrum is considerably larger than the right. The left corner of the mouth is lower than the right. Eyes: Appeared to be the same. Responded alike to light and accommodation. Vision 20/30 both eyes. Nose: Septum deviated toward right.

Left nares larger thar right. Ears: No difference noted. Tongue: Somewhat enlarged on left. Facial Muscular Activity: Apparently the same on both sides. Skin: A slightly pigmented area in the neck, extending from thyroid region toward lobe of left ear. Hair Growth: More downy hair on left lip and cheek. Scalp hairline lower in front of ear on left side of face than on right. Sweat Gland Activity: Greater on left side of face. This side perspires noticeably more than the right side in warm weather. Sensation: Normal and equal on both side sof face. No paralyses or contractures. Arms, Hands, Legs, Feet: No asymmetry could be found. Nail Growth: Same on both sides. Posture, Spine, Heart, Lungs, Abdomen, Anus and Rectum and Genitalia: Normal.

Teeth. In November, 1940: Age 9½ years (*fig. 5*). Precocious eruption on the left side, more noticeable in the upper jaw. Dental formula: $\frac{6\ V\ IV\ III\ 2\ 1\ /\ 1\ 2\ 3\ 4\ 5\ 6\ 7}{6\ V\qquad 3\ 2\ 1\ /\ 1\ 2\ 3\ 4\ 5\ 6\ 7}$ Not only was eruption much earlier on the left but /2 3 4 5 6 were unusually large teeth

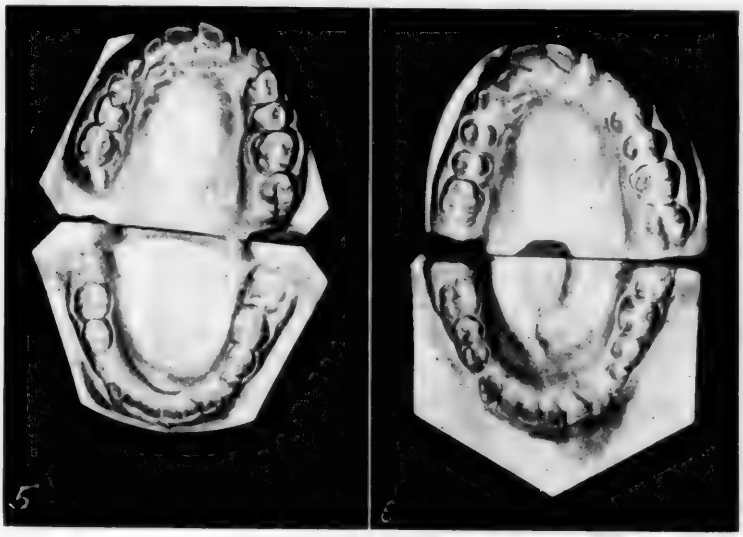


FIG. 5. Patient's teeth at age 5½ years.
FIG. 6. Patient's teeth at age 11 years.

and had erupted to such an extent that the occlusal plane on the left side was an estimated ¼ to ½ inch lower than that on the right side. Dental radiographs showed the unerupted teeth on the right side to be smaller than their erupted mates on the left. In February, 1943, at the age of approximately 11 years, gnathostatic casts (*figs. 2* and *6*) showed the following: Dental formula: $\frac{6\ 5\ 4\ 3\ 2\ 1\ /\ 1\ 2\ 3\ 4\ 5\ 6\ 7}{6\ 5\ 4\ 3\ 2\ 1\ /\ 1\ 2\ 3\ 4\ 5\ 6\ 7}$. Eruption on the right side is within normal limits for her age. Occlusal plane: Approximately ½ inch lower on left side. Midline: Deviated toward right. Size of Teeth: /2 3 4 5 6 much larger than 6 5 4 3 2/. For example: The buccal-lingual diameter of /4 is about 11.0 mm. while that of 4/ is approximately 9.0 mm. The 2 upper first incisors seem to be the same size. In the lower jaw /2 3 4 5 are noticeably larger than 5 4 3 2/ but the discrepancy is not so great as in the upper jaw. The lower first incisors seem to be the same size as also do the lower 1st permanent molars. We have no record of the comparative sizes of the deciduous teeth on the 2 sides of the jaws. A Class II malocclusion is present, accompanied by a lowering of the occlusal plane on the left side, a rotation of 2 and a linguoversion of 2/. The distocclusion

is much more pronounced on the left side. This condition and the lowered occlusal plane on the left are perhaps the result of the large size and precocious eruption of the upper teeth on that side.

Dental Caries. In November, 1940, 14 surfaces of the teeth on the left side were affected by dental caries and 3 on the right side. By June, 1943, 7 additional surfaces on the left side were carious and 1 additional on the right. To date, 21 surfaces of the teeth on the left side have been affected by caries and only 4 surfaces on the right.

Mentality. The girl is evidently normal. She is retarded a year in school due to absence caused by otitis media following scarlet fever. She has never had to repeat any of her grades. She is cheerful, friendly and cooperative.

Laboratory Findings. Nothing abnormal revealed. Urinalysis: Negative. Blood Hemoglobin: 84%. Red Blood Count: 4,400,000. White Blood Count: 7,800. Wassermann: Negative. Skin tests: Schick: Negative, Tuberculin: Negative. Basal Metabolism tests: Within normal range.

Differential Diagnosis: Congenital partial hemihypertrophy.

DISCUSSION

Various theories have been proposed with regard to the etiology of hemihypertrophy. The most common of these are: 1. Glandular dysfunction. 2. Increased blood supply to the hypertrophied area. 3. Abnormal nerve development and function. 4. Heredity. 5. Abnormal embryonic development resulting from disturbances in the germ plasm or from an imperfect form of twinning.

The evidence thus far presented in the literature gives little support to the theory of glandular dysfunction. Studies by Dana (2) and Bassoe (3) established the condition as an entity by differentiating it from acromegaly. They, however, called attention to the fact that sometimes it appeared in conjunction with acromegaly and giantism. In a case reported by Harwood and Flynn (4) a hemihypertrophy was associated with a suprarenal cortical tumor and a suggestion was made that overgrowth on one side may have been in response to growth promoting hormones produced by the tumor. Strickler (5) reported a case associated with a pituitary tumor. However, the case was believed to be an acquired rather than congenital hemihypertrophy. In the vast majority of reported cases no evidence of glandular dysfunction has been found.

That an increased blood supply to one part of the body will cause an overgrowth is well known. This increase can come about by means of a mechanical interference with venous flow, by a relaxation of nervous control over vessels in the involved area or by arteriovenous fistulae. Roubier (6) expressed belief that the etiology of hemihypertrophy is a congenital hemilateral vasomotor imbalance, with venous obstruction dependent upon fetal malposition. Chandler (7) discussed the probability of local overgrowth resulting from increased circulation of intercellular fluid or to increased O_2 tension of venous blood. In these cases, all portions involved developed at an accelerated rate of growth and the skin in these areas showed an elevation of temperature. Ward and Horton (8) stated that most cases of arteriovenous fistulas are unrecognized and are generally incorrectly diagnosed as elephantiasis, gigantism, hypertrophy, "tumor," cavernous hemangioma or varicose veins. Newer diagnostic procedures which include arteriography and the demonstration of an increased O_2 content of venous blood adjacent

to such lesions facilitate recognition of arteriovenous fistulas. They have also found that the involved part of the body has a higher temperature than the corresponding normal part and observed that bruit and thrills are generally present in acquired cases but as a rule are not present in congenital cases. The presence of nevi and hemangiomas in about half of the reported cases (9) also suggests a vascular disturbance.

The fact that about 15% of the reported cases of hemihypertrophy have been found to be associated with mental deficiency (10) suggests a disturbance in the central nervous system as an etiologic factor. The apparent increase in blood supply to the affected part also suggests that a faulty nervous mechanism may be responsible for the vasodilation. Thus far no evidence has been presented in support of these theories.

Attempts to associate hemihypertrophy with heredity have not been successful. In 1921 Gesell (10) could not find a single case wherein 2 members of the same family or related members presented hemihypertrophy. Since that time, although a few cases involving members of the same family have been reported (11, 12, 13), the fact remains that in the vast majority of cases the family history is negative. This case is therefore unusual because both the patient's mother and maternal grandmother have a noticeable facial hemihypertrophy although to a lesser degree than the patient. The record of one family, of course, offers meager evidence as to the hereditary nature of the condition. However, it does lend some support to that theory.

The theory that hemihypertrophy is an atypical or minimal form of twinning has been proposed by Gesell (9). Evidence in support of his theory is largely based upon research in experimental biology, especially that of Wilder (14), Stockard (15) and Newman (16). Gesell (9) states,

> "There can be little doubt that in some instances there is more than a morphological difference between opposite sides of the body. There is a perceptible physiologic or energetic difference which heightens the temperature on the affected side, accelerates the growth of the nails and hair and even hastens the eruption of the teeth on that side. . . . It means that sometimes an individual may have two physiologic ages at the same time, one for each half of a developmentally asymmetric body. . . . This is another striking indication of the twoness of the hemihypertrophic individual."

These statements describe the condition in the case very well. Not only is the size on one side greater but the physiological development is more advanced. If judged by the eruptive stages of the teeth on the 2 sides of her jaws, the patient would be about 2 years older on the left side of her face than on the right. Another interesting feature of this case in relation to Gesell's etiological theory is that the patient's mother is a fraternal twin. Perhaps this maternal familial tendency toward twinning lends some support to the theory that hemihypertrophy is an atypical form of twinning.

A consideration of the size of teeth may offer some valuable information. Teeth are not like bones and soft tissue whose size and contour can be modified by growth over long periods of time after birth. Teeth are unique in that their size and form are laid down early in life and are thereafter not modified. For

example, in a deciduous first incisor the size is manifested by about the fourth fetal month and nothing, in so far as we know, can bring about a further increase in size after that time. In the case of a first permanent molar the size is determined approximately by the ninth fetal month and this pattern, once outlined, cannot be enlarged. In only a few cases that have been reported have there been mentioned observed differences in the size of bilaterally comparable teeth (10, 17, 18, 19). In a case reported by Wiseberg (19) the deciduous teeth on the left side were larger than those on the right, thus the anomaly presumably originated before the fourth fetal month. In the case we are presenting, we have no record of the deciduous teeth on the left side. However, as the upper left first permanent molar is enlarged, the anomaly in all probability originated before the ninth fetal month. It has been suggested that enlargement of one side of the tongue (20) or the presence of skin markings (5) would indicate the congenital origin of a hemihypertrophy. Perhaps a more accurate record of the congenital nature could be found in the teeth. If the deciduous teeth were enlarged on one side the disturbance would be known to have occurred very early in intra-uterine life. If the first permanent molars on one side were enlarged, again it would be evident the disturbance was congenital.

The occurrence of the much greater number of dental caries in the teeth on the side of the hypertrophy could be explained on the basis of either a poorer structure of the teeth on the affected side or by the fact that because of their precocious eruption the larger teeth have been subjected to the ravages of caries for a longer period of time. Of these two explanations the first appears more probable at present, as the normal teeth on the right side which are now in place seemingly are not being attacked by caries as are those on the left, but a more definite answer to this will be available after a few years during which the teeth on the right side will have further exposure to the factors which cause caries.

From a review of the literature and a consideration of the data in this case, it would seem that congenital hemihypertrophy is embryonal in origin as suggested by Schwartzman, Grossman and Dragutsky (1). However, evidence is not conclusive whether its origin is in the germ plasm or whether it is due to early abnormal behavior of the somatic tissue. This case and its occurrence in conjunction with both a possible hereditary influence and twinning will lend support to either or both of these etiologic theories.

SUMMARY AND CONCLUSIONS

1. A case of congenital partial hemihypertrophy is presented.

2. The case is unusual in that it is the third generation in which a similar condition has appeared and in that there is also a family history of twinning.

3. The case is of orthodontic interest because of the pronounced differences in the size and eruption dates of the teeth on the two sides of the jaws.

4. We would suggest that when a hypertrophy involves the jaws, the teeth should be carefully examined. Enlargement of the teeth on the affected side definitely establishes the condition as congenital.

5. Evidence in this case points toward the disturbance as being embryonic in origin.

REFERENCES TO LITERATURE

1. Schwartzman, J., Grossman, L., and Dragutsky, D., True Total Hemihypertrophy. *Arch. Pediat.* **59:** 637, 1942.
2. Dana, C. L., Acromegaly and Giantism with Unilateral Facial Hypertrophy. *Am. Neurol. Soc. Meeting* July 26, 1893.
3. Bassoe, P. J., Gigantism and Leontiasis Ossea, with Report of the Case of the Giant Wilkins. *J. Nerv. and Ment. Dis.* **30:** 513, 1903.
4. Harwood, J. and O'Flynn, E., Specimens from a Case of Right-sided Hemihypertrophy Associated with Pubertas Praecox. *Proc. Roy. Soc. Med.* **28:** 837, 1935.
5. Strickler, F. A., Hemihypertrophy. A Case, Probably Acquired, Associated with Pituitary Tumor. *Virginia Med. Monthly* **64:** 22, 1937.
6. Roubier, C., Hemi-hypertophie Congenitale des Membres et du Pavillon de l'oreille Correspondant. *Rev. Neurol.* **27:** 147, 1920.
7. Chandler, F. A., Local Overgrowth. *J.A.M.A.* **109:** 1411, 1937.
8. Ward, C. E., and Horton, B. T., Congenital Arteriovenous Fistulas in Children. *J. Pediat.* **16:** 746, 1940.
9. Gesell, A., Hemihypertrophy and Twinning. A Further Study of the Nature of Hemihypertrophy with Report of a New Case. *Am. J. Med. Sci.* **173:** 542, 1927.
10. *Idem.*, Hemihypertrophy and Mental Defect. *Arch. Neurol. and Psychiat.* **6:** 400, 1921.
11. Reed, E. A., Congenital Total Hemihypertrophy. *Arch. Neurol. and Psychiat.* **14:** 824, 1925.
12. Scott, A. J., Hemihypertrophy. A Report of Four Cases. *J. Pediat.* **6:** 650, 1935.
13. Arnold, E. B., A Case of Hemiacromegaly. *Int. J. Orthodont.* **22:** 1228, 1936.
14. Wilder, H. H., Duplicate Twins and Double Monsters. *Am. J. Anat.* **3:** 387, 1904.
15. Stockard, C. R., Developmental Rate and Structural Expression: An Experimental Study of Twins, Double Monsters and Single Deformities, etc. *Am. J. Anat.* **28:** 115, 1921.
16. Newman, H. H., The Physiology of Twinning. (Chicago, The Univ. of Chicago Press: 1923.)
17. Werner, R., Congenital Unilateral Facial Hemihypertrophy. *Arch. f. klin. Chir.* **75:** 535, 1904.
18. Masten, M. G., Asymmetry: Unilateral Atrophy and Facial Hypertrophy. *Arch. Neur. and Psychiat.* **35:** 136, 1936.
19. Wiseberg, M., An Unusual Case of Hemigigantism. *Canad. Med. Assn. J.* **25:** 591, 1931.
20. Welt-Kakels, S., Congenital Hypertrophy of the Left Half of the Face in a Boy Seven and one-half Years of Age. *Med. Rec.* **86:** 738, 1914.

DENTAL CARIES[1]

B. GOTTLIEB

Department of Oral Pathology and Dental Research, College of Dentistry, Baylor University, Dallas, Texas

LITERATURE

Gottlieb and Stanton (1) have demonstrated that many phenomena associated with dental caries can be duplicated by the action of acid on extracted teeth. Chalky and "cloudy" enamel, cross striation of prisms, transparent enamel, dentinal shrinkage and transparent and opaque dentin were reproduced. Yellow pigmentation was not seen.

Differences of opinion concerning yellow pigmentation of dental caries are found in the literature. Abbott (2) appears to have been the first to mention it and he stated that the brown discoloration of carious tissue cannot be considered an essential feature of carious enamel. Miller (3) found more or less pronounced pigmentation in specimens of carious enamel but did not consider it an essential phenomenon of decay. Rosebury, Karshan and Foley (4) called attention to the brownish or yellowish color at the base of some molar fissures in rats. Gottlieb and E. Hinds (13) reported the presence of yellow pigment as characteristic for caries.

Gottlieb (5) showed by photomicrographs that caries can develop along a lamella into the dentin while a layer of unaffected enamel still separates the decayed enamel from the decayed dentin. He differentiated the chronically, slowly progressing form of caries, suggesting that it progresses along the narrow paths of the prism sheaths, and the acute form which attacks rapidly along the lamellae. The latter form may affect the dentin before the whole enamel is penetrated. In accordance with Baumgartner (6) and Fleischman (7) he looked at dental caries as a product of invasion of microorganisms along organic pathways. While these authors believed that invading microorganisms produce acid which causes caries, Gottlieb viewed caries as a proteolytic process. Rosebury, Karshan and Foley reported that caries might penetrate enamel diffusely, perhaps accentuating its normal structure, or that it might penetrate through a narrow lamella reaching the dentin with little apparent effect on the surrounding enamel.

E. Hinds (8) showed that ammoniacal silver nitrate spreads in the enamel like we believe that the caries process does, i.e., along organic paths not affecting the transparent, homogeneously calcified areas. Mellanby (9) stated that homogeneously calcified enamel offers a barrier against caries. Barker (10) demonstrated caries beginning along a lamella. Both the lamella and the carious

[1] Received for publication June 9, 1943.

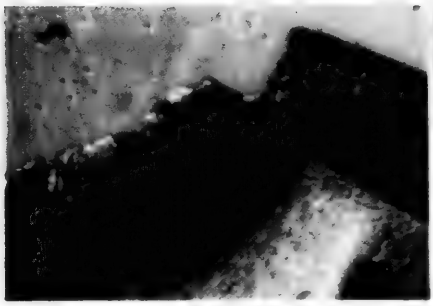


FIG. 1

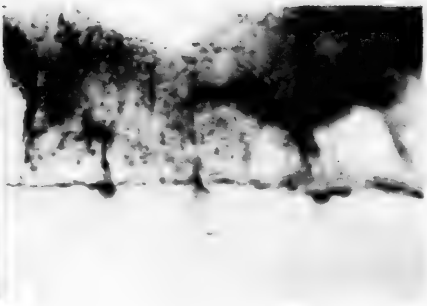


FIG. 2

FIG. 3

FIG. 4

FIG. 5

FIG. 6

FIG. 1. Yellow pigment in enamel and dentin in carious cavity

FIG. 2. Frontal caries not reaching to dentin. Three lamellae connect it with dentin. At ends of all 3 lamellae yellow spots present in dentin.

FIG. 3. Cloudy enamel, with yellow pigmentation at surface, reached dentin. The dentin here shows yellow pigmentation.

FIG. 4. Extended frontal caries reached level of tufts. In adjoining dentin yellow pigmentation with transparent dentin of first degree followed by a zone of opaque dentin.

FIG. 5. Plaque content entering lamella

FIG. 6. Yellow pigmentation at cemento-enamel junction undermining enamel from dentin

process stopped at a layer of transparent dentin. Gottlieb (11) showed that lamellae are more numerous in the approximal areas and at the thin edges of the enamel than at the buccal and lingual surfaces. The thick enamel at the greatest circumference of the teeth shows few lamellae if any are present at all. F. Hinds (12) found that caries evident in ground sections as affecting the dentin may not show up in radiographs and the enamel may be grossly intact and prevent detection by the explorer.

OBSERVATIONS

Yellow pigmentation in caries. Examination of carious cavities reveals brownish-yellow pigmentation (*fig. 1*). The edges of the enamel, as well as the dentin, show this. The surface of the dentin is dark but in the deeper areas it is lighter.

FIG. 7. Enamel lost by decalcification (a). At the connection of lamella (d) with cuticle (c) dense plaque fixed. The big loose plaque (e) has separated from the cuticle at (c).

The surface dentin apparently became contaminated by contact with the mouth cavity.

Beginning of caries by invasion of the organic pathways. *Fig. 7* is a photomicrograph of a decalcified section in which the enamel (e) has been dissolved. The cuticle with a lamella, a loose plaque and the dentin remain. The plaque appears more dense over the lamella than elsewhere. *Fig. 5* shows part of a decalcified section with a persistent, acid resistant lamella and a similarly acid resistant plaque. The basophilic content of the plaque enters the lamella and seems continuous with it. *Fig. 8* is also a reproduction of a decalcified section in which the plaque material appears continuous with the peripheral and of the lamella. The peripheral portion is thicker than the dentinal end.

Spreading of caries. A ground section through a discolored spot at the intact enamel surface is seen in *fig. 9*. Only the peripheral portion of the enamel is discolored and the zone of transverse striation (b) does not reach the dentino-

enamel junction, a strip of unaffected enamel remaining. A lamella runs to the dentin and at its point of entry the dentin is carious. In *fig. 2* the caries, charac-

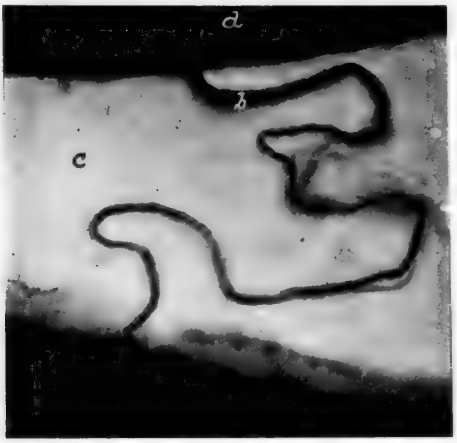


FIG. 8. Plaque (a) at surface of enamel (b) entering the lamella (c)

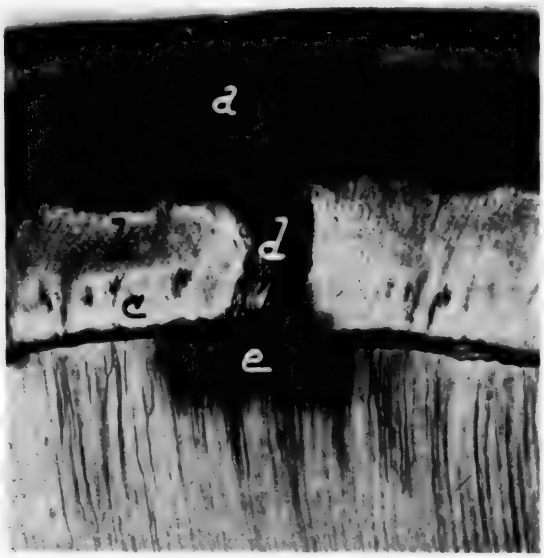


FIG. 9. Surface of enamel intact. a–carious portion; b–slightly discolored enamel; c–intact enamel; d–lamella carrying caries to dentin (e). (Reproduced from Gottlieb, Zeitschr. f. Stomat. **19:** 129, 1921).

terized by pigmentation, is spreading along the organic matter of the enamel toward the dentin. At the end of each of 3 small strands of organic material connecting the carious enamel to the dentin there is a yellow spot in the dentin.

In *fig. 3* a partly cloudy and partly pigmented process in the enamel has reached the dentin. A semicircular zone is present in the dentin at the point of contact but the surface enamel is intact. *Fig. 4* shows caries, which is quite extensive at the surface, narrowing toward the dentino-enamel junction. The surface was originally intact but a piece of enamel was broken out during the preparation of the specimen. The caries has reached the tufts but not the dentin. The tufts in the carious area are considerably thicker than those outside. The adjacent dentin has a yellow area surrounded by a lighter area which in turn is separated from the normal dentin by an opaque zone.

Fig. 6 shows an intact cemento-enamel junction with yellow pigment limited to the cementum and dentin undermining the edge of the enamel.

DISCUSSION

Seven phenomena found in enamel (chalky enamel, cloudy enamel, transverse striation, transparent enamel) and dentin (shrinkage, transparent dentin, increasing opacity) as the result of acid action in caries could be reproduced by acid action. The author does not consider a single one of these phenomena, nor all of them combined, as being caries.

Yellow pigmentation in caries. Despite all the studies of caries over a long period of time and by capable investigators the exact definition of caries is questionable. In nearly all papers in the literature chalky enamel is cited as the earliest sign of dental caries but, clinically, every chalky spot does not develop dental caries. It is known that chalky enamel is caused by acid activity but simple acid action does not produce caries *in vitro*. On the other hand every carious lesion is not chalky. A reliable criterion for indicating the presence of caries is necessary in order to provide a reliable starting point.

In advanced caries lesions, which can be reliably diagnosed clinically, ground sections show yellow pigmentation without exception. On the other hand, I do not believe that yellow pigmentation of the tooth tissue is proven attack by something other than caries. Brown stain of persistent matrix or black stain of fluoride origin must be excluded but they do not affect dentin or cementum. I do not know of any other symptom found in caries which has the two properties mentioned. Accordingly, I feel justified in drawing the conclusion that yellow pigmentation is the only and singular characteristic for the presence of dental caries.

The source of the pigment should be determined. It seems improbable that it penetrates from the surface because dentin may be pigmented beneath intact enamel and it may be found in deeper layers of the tooth when the superficial layers show none. It is assumed that the pigment is produced where it is found and not transported from another place. The pigment might be produced by necrotic material, like stagnating lymph. That shows that it is not a chemical reaction of the tissue. Invading microorganisms produce that pigment and thus caries.

Beginning of caries by invading along the organic paths. Under the same conditions a clean mouth will show less caries than an unclean one, but unclean teeth

may be immune to caries and very clean ones very susceptible. Caries does not occur under every plaque but it occasionally develops on a scoured cusp. I believe that a lamella will always be found extending from the tooth surface in each of the latter cases. In *fig. 7* a plaque is seen to become dense at the point of entry of a lamella and in *figs. 5* and *8* the plaques are seen to continue into the lamellae. In view of these findings I feel justified in stating that the invasion of certain microorganisms along the organic paths of the tooth is caries, provided these roads can be invaded. Otherwise there is natural immunity. One characteristic of these microorganisms appears to me to be their ability to produce a yellow pigment.

How the content of the plaque can enter the lamella while it is running inside the rigid walls of the enamel is not clear. A softening of the enamel prior to the invasion by acid action can be ruled out because enamel is quite hard at this time. It is probable that increase in thickness is the result of decalcification in 5% nitric acid during histological preparation. The contents of the narrow lamella expand as soon as the rigid walls are removed by decalcification. Invasion of the lamellae is far easier than invasion of the narrow crooked way of prism sheaths. Invasion is facilitated by protection of the point of entry, commonly called retention. Frontal caries, therefore, with invasion of prism sheaths is found only in protected areas, interproximally or at the gingival margins. Invasion of lamellae is easier when protection is added. Lack of cleanliness, leaving undisturbed plaques is also protection. Sometimes, for example, a premolar may rotate so that the buccal surface is in an approximal position. A lamella of the buccal surface may have escaped attack previously but in its new position with protection added caries may develop.

In 1921 (11) it was shown that the greatest circumference of a tooth has few, if any, lamellae. Lamellae appear in increasing numbers, cervically, and are found more on the buccal and lingual surfaces. At the time of that study it was pointed out that this might be related to the varying caries experiences in these areas. In young individuals the contact between the teeth is marble like (14) and approximal caries appears beneath the contact point while in older age groups the contacts are worn to planes and caries develops in these planes (15).

The cemento-enamel junction affords another point of predisposition to caries. Attack of the thin enamel edge or of the cementum is found here. At that point of the enamel the lamellae are numerous as in the thinner parts of the enamel approximally. The occurrence of caries at the newly exposed cementum appears related to natural immunity. The problem will not be discussed in detail here but the cementum and dentin have no lamella imbedded in highly calcified material which is resistant to bacterial invasion and thus to caries. These tissues are homogeneously formed and therefore susceptible to frontal caries only. Undermining caries is not found in cementum and dentin. But these tissues are less calcified than enamel (30% vs. 95% inorganic material) and are more subject to invasion. Rampant root caries can hardly be controlled. We must assume that normally a natural impregnation by the saliva (no further guess can

be offered) takes place, rendering successive belts of root resistant. Under certain unexplained conditions rampant root caries occurs while the enamel surface is resistant. It is probable that such teeth do not have impregnation of the root surface although the enamel is resistant. If such disturbances, apparently of salivary action, are limited to the saliva or are symptoms of some general metabolic disturbance I cannot even suggest.

Spreading of caries. The caries is believed by the author to spread along the organic avenues (5) and the spreading is arrested by transparent enamel (1, 10) as is the case with silver nitrate (8). *Fig. 9* shows that caries has avoided attack on the highly calcified areas of the tooth and that it has not spread into enamel surrounding the lamella. The enamel here remains intact while the dentin has a carious area at the end of the lamella. Under like conditions the dentin, containing far more organic substance, is more readily attacked by the carious process than is the more calcified enamel.

In the enamel there are 3 groups of organic structures, prism sheaths, lamellae and tufts, and all 3 are utilized by the spreading carious process. Only the prism sheaths or lamellae may be attacked from the surface. The prism sheaths are narrow and crooked and the deeper the bacteria penetrate the fewer are the opportunities for progress. For this reason caries progressing into the depth is cone shape with the tip directed toward the dentin. Such caries may be called *frontal caries* attacking the enamel in a frontal manner. Here, acid action accompanying the yellow pigmentation, forms the spearhead while the yellow pigmentation is following behind. The impression is gained that the acid producing bacteria are better able to proceed along these pathways than are those organisms that produce the pigment. The suggestion that acid and yellow pigment are produced by the same bacteria appears wrong. Both signs are found separately in frontal and undermining caries, suggesting separate sources for production of acid and yellow pigment.

The goal of penetrating microorganisms is to reach the dentin where the organic material may provide plentiful food. If they starve on the way they do not proceed and *arrested caries* results. A brownish-yellow or a dark discolored spot may remain indefinitely without further progress. If, however, frontal caries reaches the level of the tufts better roads to the dentin are afforded. In *fig. 4* the tufts appear thicker, their organic content having been enriched by the proteins of bacteria. This might indicate that the explanation given for thickening of the lamellae is incorrect. It is possible that the tufts are merely accentuated, under tension, being filled to limit with additional proteins (bacteria) but not widened. Decalcification, with removal of the rigid walls, may permit expansion. These suggestions may prove right or wrong.

Yellow pigmented areas which are more extensive than the deepest part of the affected enamel appear in the dentin. This is the *first form of undermining caries.* It also develops in instances where the tip of frontal caries reaches the dentin (*fig. 3*). If a lamella is present in the area of frontal caries, or if such a lamella had been invaded originally, the carious process progresses along the lamella and produces yellow pigmentation where it reaches the dentin. If

frontal caries was present from the start of the process the discolored area at the enamel surface is extended but limited to the superficial enamel with an intact layer separating it from the dentin. Through the intact enamel the lamella runs to the dentin where pigmentation is found. This is the *second form of undermining caries*. Cloudy enamel and transverse striation, signs of acid action, are far behind in the frontal caries area while the yellow pigmentation is forming the spearhead and has already reached the dentin. Beginning shrinkage of the dentin, a sign of acid action, is sometimes found before the transverse striation of the enamel has reached the dentino-enamel junction. This seems to indicate that acid producing organisms can progress rapidly along the lamella to the dentin before those in the prism sheaths can produce striation to the same depth. It is important to note that shrinkage follows some time after the appearance of yellow pigment, indicating that the pigment producing bacteria travel along the lamellae faster than the acidogenic organisms.

At the cemento-enamel junction a *third form of undermining caries* is found. In the course of continuous passive eruption of the tooth the base of the crevice passes the cemento-enamel junction, sooner or later. Cementum or dentin is now exposed as the surface structure and bacteria do not need to progress through the organic paths of the enamel to reach the dentin (or organically similar cementum). If conditions are favorable caries will begin immediately at the cemento-enamel junction and the highly calcified enamel will offer better resistance to dental caries than the adjoining dentin. As a result caries of the dentin at the cemento-enamel junction undermines the enamel (*fig. 6*).

A final stage of caries, which will not be discussed here, is necrosis of the enamel prisms and dentin, i.e., cavity formation.

Practical considerations. The knowledge that pigmentation is characteristic for dental caries is useful clinically. The pigment is dark brownish-gray in the mouth and if there is doubt as to the diagnosis of caries I believe we must adhere to that symptom. If the discoloration is located in a small round depression it is the discolored entrance of a lamella. As a rule such lamellae run directly to the dentin and offer a connecting pathway. If such a lesion is found no postponement of operative intervention is justified, for we can assume that the caries has already reached the dentin. Cutting into such an area we are often astonished by the extended area in the dentin while the enamel surface appears intact. The photomicrographs illustrate these conditions. If one of these areas is overlooked the pulp may be affected in 6 months, or sooner in a young individual.

In most cases shrinkage of the dentin appears to be the reason for caries showing up in radiographs. Cavity formation occurs very late through necrosis of enamel and dentin. Since caries is a proteolytic process it does not show up in the radiograph unless it is accompanied, by chance, by acid action. Acid action follows behind the most dangerous type of undermining caries where it is carried into the depth by the lamellae. For this reason radiograph evidence is expected relatively late. Isolated deepening of the surface enamel which does not penetrate the enamel is fallaciously interpreted as caries, radiographically, whereas this picture is often produced by a concave contact plane. A light area at the

dentino-enamel junction caused by dentinal shrinkage seems to be the earliest sign of caries, radiographically. It is not the author's intention to minimize the value of the radiograph in diagnosis, but to emphasize the need for immediate operative treatment if radiographic evidence of caries is present.

In some instances extended discoloration is encountered on the approximal surfaces. These represent frontal caries, but might be arrested caries which will remain as discolored spots indefinitely. However, the caries may have reached dentin and will develop into cavities in time. Postponement of cavity preparation will not do any great harm, in one instance cutting into the area may reveal carious dentin while another may show none (arrested enamel caries). If a lamella runs through a discolored area extended frontal caries may be combined with one or more areas of undermining caries (*fig. 9*). Only radiographic evidence or cavity formation will reveal this condition.

SUMMARY

1. In my opinion, the singular phenomenon characteristic of dental caries is yellow pigmentation. Its presence indicates caries, its absence excludes it. Clinically it shows up as brown discoloration and can be seen best in ground sections, better in dentin than in enamel. Chalky enamel is not carious until pigmentation appears.

2. Caries is caused by penetration of microorganisms into the organic matter of the tooth. If the narrow crooked roads of the prism sheaths are used *frontal caries* develops with cloudy enamel and transverse striated rods (signs of acid action) forming the spearhead and yellow pigmentation follows behind. If caries develops along the enamel lamellae, or if frontal caries reaches the tufts or dentin or starts at the surface of the clinical root at the cemento-enamel junction, *undermining caries* develops.

3. In my histological study there were 3 different phases observable in dental caries. The first phase is produced by acid action. The caries defining, apparently proteolytic phase is characterized by production of yellow pigment in the dental tissues while they still retain their tissue character. The third is the necrotizing phase in which the tissue is turned into necrotic debris resulting in cavity formation. Histologically it could not be decided if it is a separate phase or an advanced stage of the second phase. The separate appearance of these phases, histologically, suggests their independence with separate bacteriological backgrounds.

REFERENCES

1. Gottlieb, B., and Stanton, S., *Texas D. J.*, **61**: 293, 1943.
2. Abbott, F., *D. Cosmos*, **21**: 57, 1879.
3. Miller, W. D., *Microorganisms of the mouth* (Phila., S. S. White: 1890).
4. Rosebury, T., Karshan, M., and Foley, G., *J. D. Res.*, **13**: 379, 1933.
5. Gottlieb, B., *Zeitschr. f. Stomat.*, **19**: 129, 1921.
6. Baumgartner, E., *D. Monat. f. Zahnh.*, **29**: 321, 1911.
7. Fleischman, L., *D. Cosmos*, **56**: 1397, 1914.
8. Hinds, E. C., *J. D. Res.*, **21**: 475, 1942.
9. Mellanby, M., *J. A. D. A.*, **17**: 1456, 1930.

10. Barker, F. J., *J. A. D. A.*, **18:** 17, 1931.
11. Gottlieb, B., Ost.-Ung. 1/4 Jahrschr. Zahnh., **31:** 19, 1915.
12. Hinds, F. W., *J. A. D. A.*, **30:** 257, 1943.
13. Gottlieb, B., and Hinds, E. C., *J. D. Res.*, **21:** 317, 1942.
14. Black, G. V., *Pathology of Hard Tissues of Teeth* revised by A. D. Black (Chicago: Medico-dental Publ. Co.: 1926).
15. Beust, T., *J. D. Res.*, **15:** 453, 1935–36.

INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

Scientific Proceedings of the New York Section[1]

January 27, 1944[2]

Compiled by Frances Krasnow, Ph.D., Editor, New York Section

Guggenheim Dental Clinic, New York, N. Y.

1. Radio-active studies on dentin. *Gerrit Bevelander, Ph.D.*, and *Melvin Amler, A.B.**, College of Dentistry, New York University, New York. These studies are being made on human teeth to ascertain the relative degree of permeability of radio-active phosphorus in different morphological varieties and different topographical areas of dentin. The procedure consists of sectioning teeth on an abrasive wheel to a degree which allows the tissues to be recognized readily by means of the microscope. Then the sections are immersed in a solution of radio-active phosphorus (P^{32}) for a suitable period. The desired tissue or parts of tissue are identified, separated, finely powdered and weighed. Samples of several teeth are pooled for each measurement. The activity of the various samples of dentin were ascertained by means of measurements made with either an electroscope or a Geiger-Muller counter. Our results thus far indicate the dentin in the circumpulpal region to be much more permeable than any other—apical dentin slightly more than dentin in the coronal region. When measurements were made on the basis of morphology the following ratios were observed: circumpulpal: 54.6; coronal: 13.3; apical: 16.5; adventitious (secondary): 20.9. It is thus apparent from the above data that different topographical areas of the teeth reveal differences in the rate of the amount of permeability of phosphorus in the activated form. These differences are still more striking when the measurements are correlated with the morphology of the tissue itself.

2. Simple demonstration showing action of local anaesthetic bases in comparison to that of their salts. *Isaac Neuwirth, Ph.D.*, College of Dentistry, New York University, New York. Investigations (reported in the literature) using plants and tadpoles have demonstrated that the salts of alkaloids are much less toxic than are the free alkaloids. Along similar lines, it has been found that the local anaesthetic bases, cocaine, procaine, eucaine, etc. had a stronger anaesthetic action on tadpoles, on nerve fibers and when injected intradermally, than had their salts. In other words, it is the free base that is

[1] The cost of publication of these proceedings is paid by the New York Section. Received for publication January 31, 1944.

[2] Presiding—John Oppie McCall, Guggenheim Dental Clinic.

* Guest.

the active agent, a finding in line with the Meyer-Overton theory of lipoid solubility and narcosis. Recently goldfish have been used to study the efficiency of local anaesthetics. In such a study, it was shown that cocaine is ten times as efficient as procaine, a finding which is now accepted as being a measure of relative toxicity of these two substances. As a laboratory demonstration, we have been using goldfish not only to indicate the relative toxicities of cocaine and procaine, but by placing the salts of these local anesthetics in a weakly alkaline solution (50 mgm. sodium carbonate per liter) to show also that the free base is the active agent in producing the effects of these local anaesthetics. The demonstration is carried out using six 600 cc beakers, each containing 250 cc of solution and 2 goldfish. The controls and test solutions are distributed among these six beakers as follows: 1. Distilled water; 2. Sodium carbonate solution, 1:20,000; 3. Procaine hydrochloride solution, 1:500; 4. Cocaine hydrochloride solution, 1:5000; 5. Procaine hydrochloride 1:500 in the sodium carbonate solution; 6. Cocaine hydrochloride 1:5000 in the sodium carbonate solution. The fish in the last two beakers die quickly within two minutes, those in beakers 1 and 2 are not affected at all, while those in beakers 3 and 4 may not be affected for 15 minutes or more.

3. MOUTH CONDITIONS IN RATS UNDER A LOW PANTOTHENIC DIET WITH THE ADDITION OF ZINC CARBONATE. *Daniel E. Ziskin, D.D.S., George Stein, M.D., D.M.D., Paul Gross, M.D.**, and *Edith Runne, A.B., M.A.**, School of Dental and Oral Surgery, Columbia University, New York. Examination was made of the heads of rats fed a synthetic diet complete in every respect but suboptimal in pantothenic acid and containing 0.8% zinc as zinc carbonate. This method of demonstrating existence of a toxic effect of zinc by feeding a suboptimal synthetic diet was first used by one of us (P. G.). The diet was started at about the age of weaning and the rats were sacrificed, or died, at the ages of 11 to 14 weeks. Although the animals developed the clinical features of pantothenic acid deficiency, the condition was precipitated by the zinc poisoning as was demonstrated by the control animals maintained on identical diets without zinc carbonate. The resultant skin lesions consisted of caking of the whiskers; crusting about the eyelids and spectacled eyes; crusting of the nose; rusting of the fur; and alopecia of varying degree. The mouth lesions encountered were fissures and crusting at the angles, and ulcers on the mucous membranes of the cheeks and tongue. The ulcers often started as subepithelial vesicles surrounded by necrosis involving the germinal layers of the epithelium and the papillary layer in the corium. The necrosis became narrower as it reached the surface and was granular in appearance. In the early stage, practically no inflammatory reaction was seen in the corium but appeared later, strikingly mild in intensity and small in amount as compared with other types of ulcers. Soft deposits accumulated on the incisors. In many, the incisors were depigmentized and clinically resembled the teeth in fluorosis, avitaminosis A and E and in anemia. Features of zinc poisoning, not found ordinarily in pantothenic acid deficiency

* Guest.

in rats, were mouth ulcers, decolorization of the teeth and anemia. When optimal amounts of pantothenic acid were supplied to animals fed a diet mixture containing 0.8% zinc, the skin and mucous membrane lesions described above did not occur. These experiments demonstrated the toxic effects of large doses of zinc carbonate and a protective mechanism involving pantothenic acid.

4. INCIPIENT ORTHODONTIC LESIONS AND THEIR TREATMENT. *Carl Breitner, M.D., D.D.S.*, New York. While the improvement of orthodontic appliances may still form an objective of orthodontic progress, research dealing with biomechanics seems to point a way toward their reduction or elimination. A better understanding of the inherited pattern of development upon which environmental influences are superimposed promises greater success in the prophylaxis and treatment of dento-facial anomalies. The causes of such anomalies are chiefly two-fold: (1) damaging environmental influences; (2) disturbances in the developmental mechanics. Orthodontic prophylaxis has so far concentrated upon the elimination of the damaging environmental influences; for our present knowledge is insufficient to attempt modification of the inherited pattern of development in practice. It seems, however, that the great majority of disturbances of this variety is not permanent but temporary. There exist two developmental processes, occuring simultaneously, but independent of each other, which must be perfectly correlated in time and space in order to create a normal masticatory apparatus. In such a complicated arrangement temporary disturbances may frequently occur. While in other organs such temporary disturbances may straighten out spontaneously, it is characteristic of the dentition that a temporary disturbance usually becomes permanent through mechanical fixation. This explains the great number of malocclusions in mankind, and it also points to successful self-correction of such malocclusions. Such a way is the elimination of any mechanical interference which hinders normal development. Clinical evidence shows that, for instance, cases of Angle's Class II which exist at an age of between eight and ten and which certainly would persist throughout life unless interfered with, heal spontaneously if unimpeded growth of the mandible is permitted through elimination of mechanical hindrances. It is evident that such measures as are advocated in this report are to be instituted during the period of accelerated growth, that is, between the ages of five and eleven years.

5. A TEN YEAR STUDY OF A CASE OF OSTEOFIBROSIS. *Sidney Sorrin, D.D.S.*, College of Dentistry, New York University, New York. This study consists of a most interesting case of osteofibrosis which has been under observation for a period of ten years. During this time, there had been a progressive filling-in of bone in areas of rarefaction. Recent roentgenographic series indicate complete eradication of the osteofibrosis. Systemic examination from time to time revealed no pathosis. It is evident that the result was due entirely to the elimination of occlusal trauma.